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THE INCIDENCE OF ALCOHOLIC PSYCHOSES IN MASSACHUSETTS, 1917-1935*

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THERE is probably no single subject in the history of medicine producing so many different opinions as alcoholism. Emotionally motivated arguments on its varied aspects have been known to occur even in scientific circles. One point is quite clear, however. Physicians and psychiatrists owe to society their most serious efforts to curb this disorder. Alcoholism and venereal diseases, so often found associated, have tremendous social and economic implications and are extremely important from the point of view of public health. Preventive medicine has long recognized the need for intensification of the present programs of education of the general public in particular reference to the effects of alcohol. Professional interest and consideration are being directed to the symptomatology, pathology and treatment of alcoholism. The psychopathology of this disorder is receiving appropriate attention also.[1, 2] However, the epidemiology of alcoholism has never received adequate attention at the hands of the medical profession.

Physicians in general, and psychiatrists in particular, are best qualified to minister to the needs of the alcoholic patient. Their interest should not cease when delirium or hallucinations have subsided.[3] The personal and sociologic problems of these individuals are continuous and not only influence the health of the patient but the general well-being of the community.[4] To attempt to deal with the alcoholic patient on an impersonal basis is more or less a waste of time. The social setting which encourages the formation of alcoholic habits deserves scientific approach as well as the study of the personality make-up of the individual. The problems of the alcoholic patient are not confined entirely to his own personality. He lives in a large social group and should not be treated as an isolated unit. His influence upon the community and, more especially, its influence upon him deserve serious consideration.[5] From many points of view, therefore, it will be enlightening to review the recent history of alcoholism in this State and see what progress is being made.

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PURPOSE

Alcoholism, and its aftermath in the mental field, should be included in the list of controllable and preventable disorders. Through adoption of the Eighteenth Amendment, the United States has witnessed a notable effort in the direction of control of this disorder over the past twenty years. The efficiency of this approach may be tested by studying its effect upon the incidence of the alcoholic psychoses in the mental hospitals of Massachusetts.

The following presentation will show the relation between prohibition and the incidence of the alcoholic psychoses in both metropolitan Boston‡ and in the remainder of the State over the period 1917-1935. What are the effects of restriction in the consumption of alcohol in the case of city inhabitants as contrasted with the residents of smaller communities? What are the age factors in reference to the alcoholic psychoses? What has been the effect of the Eighteenth Amendment on associated medical and social factors over the period 1917-1935? It is hoped that the data presented may add to the knowledge and understanding of what has gone before and what is now happening in this Commonwealth with particular reference to the influence of alcoholism on the psychoses, and other related factors.

THE ALCOHOLIC PSYCHOSES IN MASSACHUSETTS, 1917-1935

Graph I presents the first admissions with alcoholic psychoses from the metropolitan Boston area§ and from the remainder of the State,‖

‡Nearly one half of the population of Massachusetts is found within the area known as metropolitan Boston. Cities and towns included are as follows:—Arlington, Belmont, Boston, Braintree, Brookline, Cambridge, Canton, Chelsea, Cohasset, Dedham, Dover, Everett, Hingham, Hull, Lexington, Lynn, Malden, Medford, Melrose, Milton, Nahant, Needham, Newton, Norwood, Quincy, Reading, Revere, Saugus, Somerville, Stoneham, Stoughton, Swampscott, Wakefield, Walpole, Waltham, Watertown, Wellesley, Weston, Westwood, Weymouth, Winchester, Winthrop, Woburn. The Decennial Census, 1935, Secretary of State, Massachusetts, table 10, page 23.

§Admissions diagnosed as alcoholic psychoses to the Boston Psychopathic Hospital and the Boston State Hospital. Transfers are not considered. This excludes the Metropolitan State Hospital, which, up to the present date, has been receiving Boston cases resident in other mental hospitals, as transfers. The admission rates for Boston are based upon the population of the metropolitan area, aged 20 years and over.

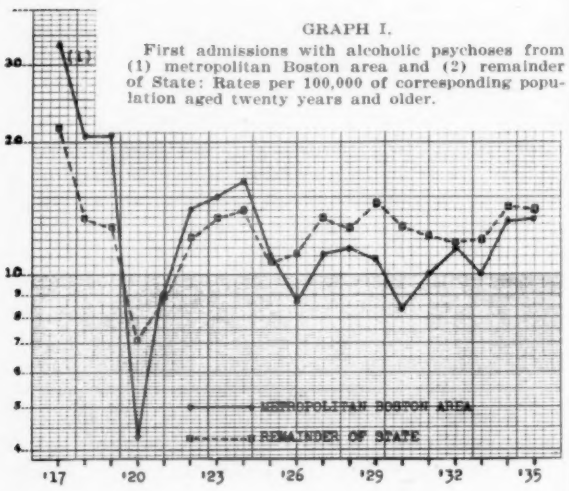
‖Admissions diagnosed as alcoholic psychoses to the Danvers, Foxboro, Gardner, Grafton, Medfield, Northampton, Taunton, Westboro, Worcester, Monson, and Bridgewater State Hospitals; Mental Wards at the State Infirmary; and Veterans Facilities No. 95 and No 107. Admission rates are based upon the entire State population, 20 years and over, *minus* the metropolitan area.

over the period 1917-1935. First admissions from the metropolitan Boston area, as shown by the heavy line, present the high rate in 1917. This group shows a decrease in 1918 and 1919, and a marked drop in 1920 to the low point for all years. Higher rates are observed from 1922 onward, with 1922-1924 at a higher level than the succeeding years 1925-1933. The years 1934 and 1935 show increases which are definitely above the rates of 1925-1933 but below those of 1922-1925 and the preprohibition years of 1917-1919.

First admissions from the remainder of the State (the dotted line) are lower than those from the metropolitan area in 1917-1919, and

GRAPH I.

First admissions with alcoholic psychoses from (1) metropolitan Boston area and (2) remainder of State: Rates per 100,000 of corresponding population aged twenty years and older.



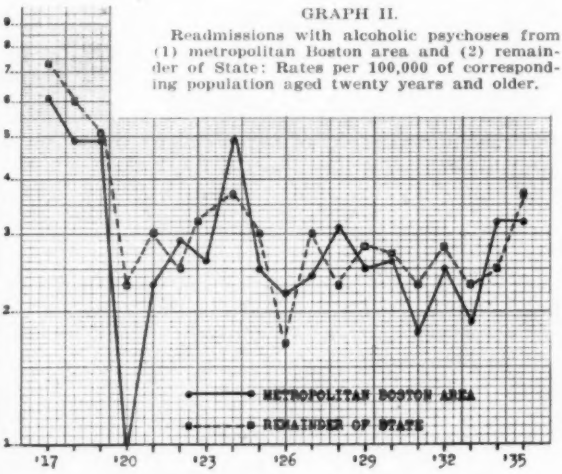
then show the same extreme drop in the first year of the Eighteenth Amendment, 1920. From 1922 onward the rates for the rest of the State remain on the same general level. From 1926 to 1931 the rates for the Boston area drop below those of the remainder of the State. From 1932 to 1935 the curves for the two areas draw closer together. In the six-year period 1926-1931 the metropolitan area makes a better showing in the alcoholic psychoses than the cities, towns and villages in the remainder of the State. Nineteen thirty-four and 1935 show rises in both groups from the rates of 1933. However these rates do not depart greatly from those of the earlier years of prohibition, 1922-1924.

Graph II presents the same data for readmissions. The heavy line representing the metropolitan Boston readmissions shows very high rates up to 1919, a precipitous drop in 1920, a rise to a second high in 1924 and from that point onward fluctuations tending to slightly lower levels up to and including the year 1933. For the repeal years of 1934-1935, we note a sharp rise which is above the level of the prohibition years with the exception of 1924. Readmissions from the remainder of the State were slightly higher than the Boston area up to 1919, but do not show so great a drop in connection with 1920, the first year of the Eighteenth Amendment. From 1920 on, the readmissions from the rest of the State show a high year in 1924 and a low in 1926, otherwise the fluctuations remain practically on the same level without the downward trend noticed in the Boston readmissions. In the repeal years of 1934 and 1935 readmissions from Boston reacted immediately with higher rates. The remainder of the State did not react so quickly, however, showing little change in 1934, followed by a substantial increase in 1935.

In both first and readmissions we have observed that the prohibition years present a general level of admission rates which were lower than the rates for the preprohibition years, 1917, 1918, and 1919. The first year of the Eighteenth Amendment, 1920, demonstrated admis-

GRAPH II.

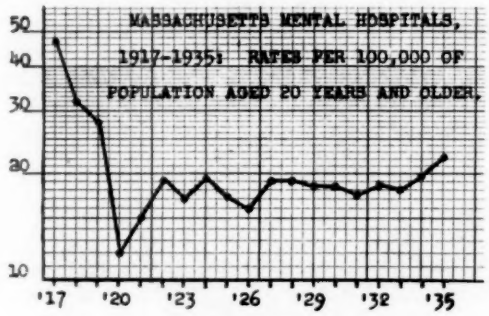
Readmissions with alcoholic psychoses from (1) metropolitan Boston area and (2) remainder of State: Rates per 100,000 of corresponding population aged twenty years and older.



sion rates which were only one fifth to one half as high as those of the preceding years. Admissions from the metropolitan area showed a more marked reaction to the imposed restrictions of 1920, with lower rates after the high rates of 1922-1924. The remainder of the State did not follow this same course but shows rather flat curves from 1922 onward.

Graph III shows the rates for first admissions classified as "intemperate" in the use of alcohol, to Massachusetts mental hospitals, 1917-1935. These are not cases with alcoholic psychoses exclusively, but include patients with other types of psychoses who were known to be intemperate in the use of alcohol. This material shows the significance of alcoholism as a possible etiologic factor in psychoses other than those of purely alcoholic origin. The years 1917, 1918,

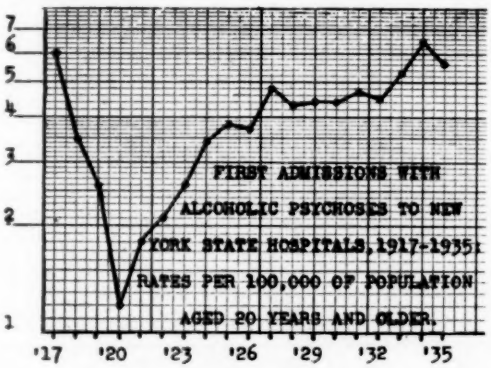
and 1919 present high rates. The year 1920 shows the lowest rate for the nineteen-year period. There is a rise for 1921 and 1922, but the high points of the preprohibition years are not approached at any time. The curve remains essentially on the same level throughout prohibition, but the repeal years of 1934 and 1935 are somewhat higher. The period of the Eighteenth Amendment, 1920-1933, presents decidedly lower rates for intemperate alcoholic habits than the years 1917, 1918, and 1919.



GRAPH III.

First admissions classified as "Intemperate" in the use of alcohol.

As a check on the Massachusetts figures data on first admissions with alcoholic psychoses in New York State Hospitals over the same years are presented (Graph IV). New York has had



GRAPH IV.

the same general experience as Massachusetts. The year 1917 is high. The first year of prohibition, 1920, presents the lowest rate of all years. In New York the immediate reaction following 1920 was not so great. Massachusetts showed a quick rise from 1920 and tended to remain on the same level or to show decreases from 1922 onward. New York presents a steadily rising curve which does not begin to flatten out until 1926. The New York figure for 1934 surpasses the high rate of 1917. Judging from the incidence of the alcoholic psychoses, Massachusetts made a better showing in the control of alcoholism during both the prohibition and repeal years.

AGE TRENDS IN THE ALCOHOLIC PSYCHOSES IN MASSACHUSETTS, 1917-1935

The age of first admissions* with alcoholic psychoses is studied in Graph V. It will be noted that the age group 40-49 years presents the highest general admission rates. The 50-59 year age group is slightly lower, followed by the 60-69 group and the 30-39 year group. The lowest admission rates occur in the 20-29 year group.

The various age groups show different reactions to the withdrawal of liquor in 1920. Those between 20 and 49 show rather remarkable drops in the year 1920. Those from 50 to 69 show a lesser reaction, while the 70-79 year age group shows no drop in 1920. The reaction in the younger ages and the lesser reaction in the older ages to the restrictions of 1920 suggest that alcoholic psychoses of the younger ages, which have had a shorter period of drinking in the background, are more responsive to the withdrawal of alcohol. In the younger ages the incidence drops quite markedly in 1920 when alcohol was withdrawn. Conversely, the alcoholic psychoses of the older ages have many more years of drinking preceding the psychosis. They present, in addition, combinations of personality deviations with senile and arteriosclerotic changes. Apparently the resulting mixtures of disorders are more chronic in nature and less dependent upon alcohol or changes in the drinking habits.

Most interesting of all are the various age trends throughout the prohibition era. Temporarily discarding the rising rates for the repeal years of 1934 and 1935 and confining our observations to the prohibition years between 1922 and 1933, we note unmistakably decreasing trends in the 20-29, 30-39 and 40-49 year age groups. The 50-59 year age group shows a slight upward trend. The 60-69 and 70-79 year groups present even more definite upward tendencies. In other words, the alcoholic psychoses (first admissions) are moving out of the younger age groups and into the older age groups.† Most encouraging of all is the marked downward trend in the younger ages between 20 and 29 years. The repeal years of 1934 and 1935 present increases in rates for the ages under 40, but only slight changes between 40 and 70.

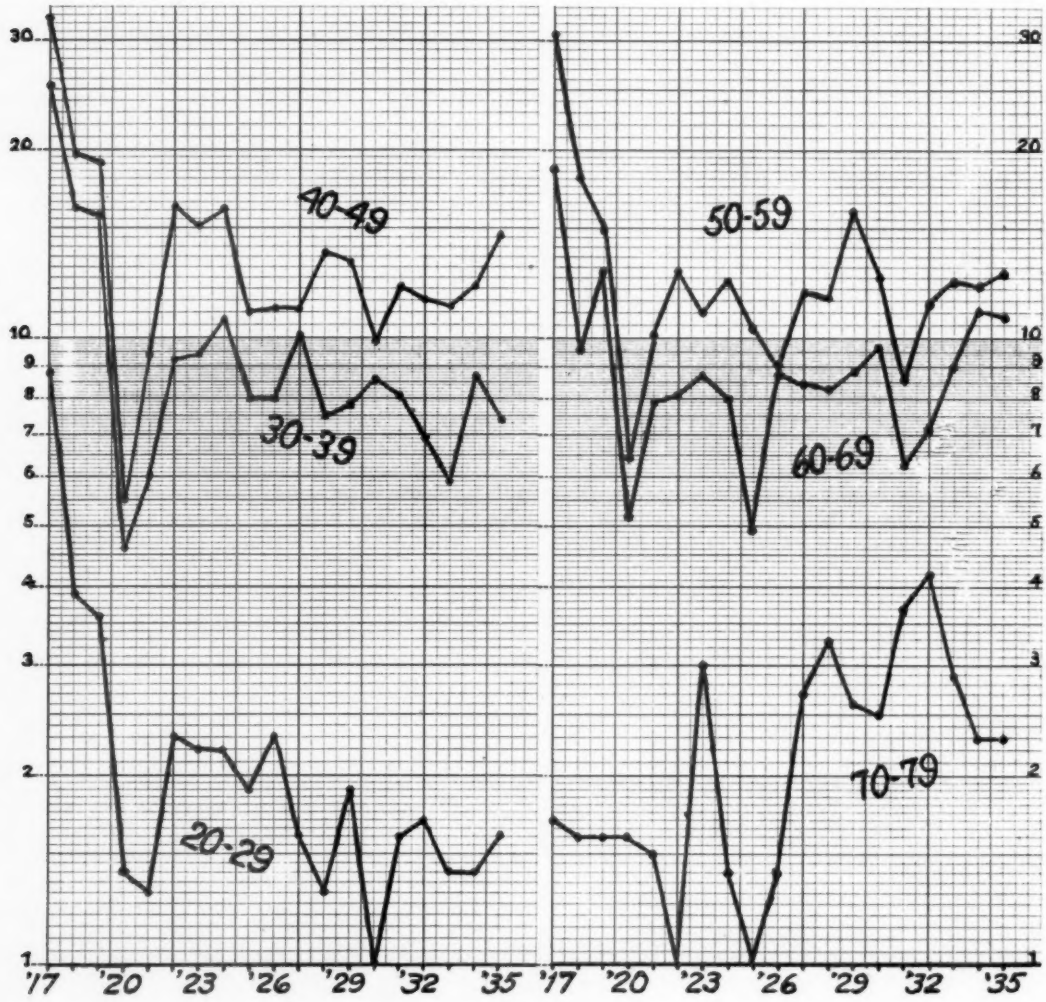
DRUNKENNESS IN MASSACHUSETTS, 1917-1935

Up to this point we have confined ourselves almost entirely to the incidence of the alcoholic psychoses. Let us now add data from related fields to test whether the observed changes during the prohibition era are associated solely with

*First admissions within each age group for each year are compared with the population figures for the same ages and years.

†The observed changes in age rates are, of course, quite independent of the well-known shift of our population to the older ages. The aging tendencies of the alcoholic psychoses are similar to, but decidedly in excess of, those observed in the general population.

mental disorders. Graph VI shows the population rates on arrests for drunkenness in Massachusetts over the period 1917-1935.† High rates for drunkenness prevailed in 1917, 1918 and 1919, with a precipitate drop to the low point for all years in 1920. Rises occurred in 1921 and 1922. The high points of the prohibition era occur in 1923 and 1924. The arrests for drunkenness then decrease to a second low point in 1932. The year 1933 shows a slight rise, and the repeal years of 1934 and 1935 show rates above those of the prohibition years 1920-1933. The general contour of this curve is very similar to that of Graph I, showing the first admissions with alcoholic psychoses.



GRAPH V.

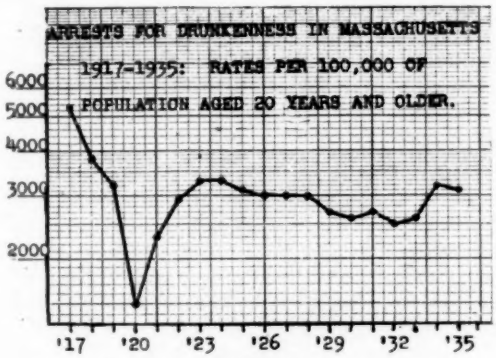
Age of first court admissions with alcoholic psychoses, all Massachusetts Mental Hospitals, 1917-1935: Rates per 100,000 of corresponding population.

†Acknowledgment is made to the Massachusetts State Department of Correction for furnishing the data upon which these rates were based.

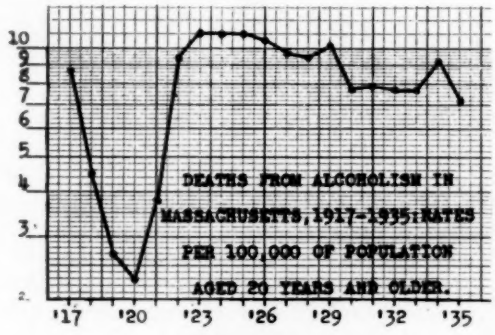
DEATHS FROM ALCOHOLISM IN MASSACHUSETTS, 1917-1935

Graph VII shows the deaths from acute and chronic alcoholism in Massachusetts over the years 1917-1935.[6] The obviously high rates during the years 1922-1933 demonstrate one of the least favorable aspects of the prohibition era. Eight years of the prohibition period, 1922-1929 inclusive, present death rates which are higher than those of 1917. The extremely inferior or even poisonous quality of many of the beverages offered for sale during the early years undoubtedly influenced the high rates observed, particularly from 1922 to 1926. From 1926 to 1933 the quality of illegal beverages improved and the death rates from alcoholism fell to lower levels.

Although the death rates during the prohibition era seem high, it should be recalled that they involve only between 59 (1920) and 300 (1925) deaths per year out of a total population of over 4,000,000 persons. Even in these years, alcoholism could not be considered as other than a minor cause of death. For instance, in the high year of 1925, a total of 300 deaths were involved. That year alcoholism was twenty-fourth in numerical order of the various causes of death in Massachusetts.

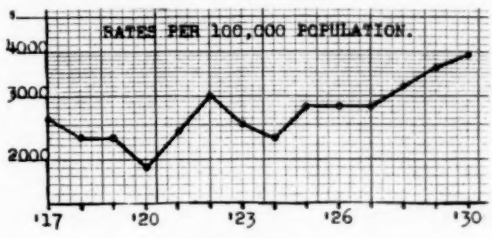


GRAPH VI.



GRAPH VII.

In Graph VIII we turn to the social field and show population rates for the number of persons supported or relieved by cities and towns in Massachusetts during the period 1917-1930.[7] In 1917, 98,000 persons were assisted in Massachusetts. In 1918 and 1919 the number reported was 89,000 for each year. In 1920 there was a drop to 75,000 persons, or 14,000 fewer than in the two preceding years. For 1921 and 1922 we see marked increases, 1922 with 121,000 rising above those of the preprohibition years. The year 1924 shows a rather marked drop to 95,000. From this year onward there is an increasing trend. The high figures for the years beyond 1930 have not been included since certain elements enter here in connection with the depression. One point is outstanding, however. Nineteen twenty, the year showing the low rates in the alcoholic psychoses, in drunkenness, in alcoholic deaths, and so forth, also shows the low point for public charity in Massachusetts. In considering these facts it should be remembered that 1920 was not the most favorable year of the postwar boom, as higher incomes were reported in Massachusetts in both 1918 and 1919. The Federal income taxes from personal returns



GRAPH VIII.

Persons supported or relieved during the year by cities and towns of Massachusetts, 1917-1930.

reported collections of eighty-one million dollars in 1918, eighty-six millions in 1919, sixty-nine millions in 1920, and smaller amounts in the succeeding years.[8]

DISCUSSION

For convenience in analysis, the prohibition and the repeal years of 1920-1935 are divided into four periods. Each period marks a fairly well-defined change in the drinking habits of the citizens of this Commonwealth. The year groups are as follows: (A) 1920, (B) 1921-1924, (C) 1925-1933, and (D) 1934-1935.

In the first period, A, but one year is placed, 1920. This year shows the full effect of the first twelve months of prohibition. The emergency prohibition of the latter half of 1919 had already started the preliminary work in cutting down the consumption of liquor. With the legal sources of manufacture and distribution rendered inoperative and illegal traffic as yet undeveloped, 1920 produced the nearest approach to real prohibition that this country has ever experienced.

What effect accompanied this first year of the Eighteenth Amendment? The alcoholic psychoses (both first and readmissions) from the metropolitan Boston area and the remainder of the State showed tremendous drops to admission rates which were only one half to one fifth of those observed in the preceding years. As far as the effect in the various age groups is concerned, the decreases in admission rates were more marked under the age of 40 and less marked between the ages of 40 and 70. In 1920 arrests for drunkenness, deaths from alcoholism, State Hospital admissions with alcoholic psychoses in New York State, intemperance in mental patients not diagnosed as alcoholic psychosis in Massachusetts, and number of persons receiving public charity in Massachusetts, all show very marked drops to the lowest rates of all the years under consideration. In the changes re-

corded in the medical, psychiatric and social fields, the year 1920 is unique in the history of this Commonwealth.

The second or B phase of the prohibition era includes the years 1921-1924. During these years the consumption of alcohol on the part of the great mass of occasional or moderate drinkers was reduced materially. The chronic drinkers, who constituted a much smaller proportion of the drinking population, continued to seek anything that contained alcohol. Poisoning from methyl alcohol and from the drinking of the various alcoholic preparations became common. This period shows the most formidable statistics of the prohibition era, with deaths from alcoholism and arrests for drunkenness rising to higher levels. First admissions with alcoholic psychoses in both the metropolitan Boston area and the remainder of the State showed higher rates in 1922, 1923, and 1924, than in the years following. Readmissions throughout the State showed higher rates during these years also. Persons requiring relief show a sharp increase during the years of this second period, rising from 75,000 in 1920 to 121,000 in 1922. The numbers then drop to a lower level, the figure of 1922 not being surpassed until the year 1928 when 133,000 persons required public relief.

The third subdivision of the prohibition era, C, extends from 1925 to 1933. While occasional poisonings continued, the general quality of the liquor offered was better. Drinking probably increased on the part of many individuals who could not be tempted by the questionable beverages offered during the earlier years of prohibition. The C period, 1925-1933, provides an apparent paradox. Drinking probably increased and yet corresponding increases are not observed in the various other factors considered in this study. First admissions and readmissions with alcoholic psychoses from the metropolitan Boston area were decidedly lower during this period than in the four years immediately preceding. In the remainder of the State the rates stayed at about the same level. Deaths from alcoholism, which were very high in the B period, 1921-1924, show decreases to lower levels, the low point being reached in 1932. The decreasing rates for alcoholic deaths during this third period point toward the quality of the liquor as a significant factor in the high death rates of the years 1921-1924. Arrests for drunkenness also show a tendency to lower levels beginning with 1925. The drunkenness rates drop to a second low in 1932 which matches the second low in the death rates from alcoholism.

The fourth or D period includes 1934 and 1935, the years immediately following repeal of the Eighteenth Amendment which became operative on December 5, 1933. During these years beer, wine and liquors of good quality became available immediately. First admissions with alcoholic psychoses in Massachusetts show immediate increases, the metropolitan area increasing from 10 per 100,000 in 1933 to 13 per 100,000 in 1934. First admissions from the remainder of the State increased from 12 to 14. The rates for 1935 remained on approximately the same level as in 1934. In the readmissions the metropolitan Boston area showed an increase from 1.9 in 1933 to 3.2 in 1934, with 1935 on the same level. The readmissions from the remainder of the State showed a small increase from 2.3 in 1933 to 2.5 in 1934, and a larger increase to 3.7 in 1935. In New York State we find the 1934 rates running to a higher level than the peak year of 1917. Massachusetts first admissions classified as intemperate in the use of alcohol showed increases to higher rates than any of the prohibition years, exceeded only by the years 1917, 1918 and 1919. Deaths from alcoholism show a rise from 7.6 in 1933 to 9.2 per 100,000 in 1934, and a drop to 7.2 in 1935. Arrests for drunkenness show a sharp increase from 2,600 per 100,000 in 1933 to 3,200 in 1934 and 3,100 in 1935.

One point is invariably raised in rebuttal of the good effects observed during the first year of prohibition, 1920. It is stated that a downward trend in alcoholic habits was already in operation in the years 1918 and 1919 before the Eighteenth Amendment went into effect. In other words, the year 1920 merely registers a continuation of the existing trend. This reasoning fails to give consideration to the very important series of events occurring in 1917, 1918 and 1919. Several factors became operative during the war years which tended to reduce the consumption of alcohol very materially. Restrictions affecting the Army and Navy personnel went into effect in May, 1917.[9] Massachusetts had nearly 200,000 men in Government Service. Thus, approximately 10 per cent of the male population was subjected to rigid restrictions in the consumption of alcohol. The Food Control Act forbidding the use of foodstuffs, grains and so forth, for the manufacture of liquor became operative in August, 1917.[10] The Zoning Act prohibiting the sale of liquors in areas surrounding mines, shipyards, war industries, and so forth, was passed in September, 1918.[11] Finally war-time prohibition pending the termination of demobilization[12] was passed and all sale of liquor prohibited after June 30, 1919. In addition, local option and other restricting influences had continued to gain during the war period. Each of these elements acting in conjunction had an effect in reducing drinking in 1918 and 1919 and in diminishing other consequences associated with the long-continued consumption of alcohol.

SUMMARY

1. A rather remarkable showing was made in the year 1920, the first year of the Eighteenth Amendment. During that year we find (a) low admission rates for first and readmissions with alcoholic psychoses, both from the metropolitan Boston area and the remainder of the State; (b) low rates in first admissions classified as "intemperate" in the use of alcohol; (c) low rates in alcoholic psychoses admitted to New York mental hospitals; (d) low rates in arrests for drunkenness in Massachusetts; (e) low rates in deaths from alcoholism in this State; and (f) low rates in the number of persons supported by public charity in this Commonwealth.

2. Following 1920 the alcoholic psychoses, arrests for drunkenness, and intemperate habits in all first admissions of mental cases rise to, and remain on, higher levels throughout the years 1921-1933. At no time, however, do they approach the levels of the preprohibition years, 1917, 1918, and 1919.

3. Decreasing rates during the latter period of prohibition, 1925-1933, are observed in the alcoholic psychoses, in arrests for drunkenness, and in deaths from alcoholism.

4. Rises in the alcoholic psychoses, in arrests for drunkenness, and in alcoholic deaths occur with the first repeal year, 1934. Studies of subsequent years will be necessary, of course, to determine whether these changes in trend are possibly chance fluctuations.

5. Within the prohibition era several factors present decreasing trends from the year 1922 to 1932 and 1933. Apparently some adjustment was going on during these years making for lower admission rates in the alcoholic psychoses, fewer arrests for drunkenness, and fewer deaths from alcoholism. Repeal, however, changed this trend and started these factors in the upward direction. However, it should be noted that, in the license years of 1934 and 1935, conditions were better than they were in the earlier years of license before 1920.

6. Throughout the years of the Eighteenth Amendment there was an apparent drift in the alcoholic psychoses from the younger age groups to the older. In the 20-29 year age group the decreasing trend is most pronounced and persists in both the 30-39 and 40-49 year age groups. Above 50 years the alcoholic psychoses show increases, more marked above 60 years. The younger age groups with a decreasing incidence during prohibition show a reversal of trend and upward tendencies in the repeal years of 1934 and 1935.

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SPONTANEOUS INTERNAL BILIARY FISTULAS*

A Review and Report of Two Cases

BY WILLIAM S. ALTMAN, M.D.,† AND EUGENE A. FIELD, M.D.†

SPONTANEOUS fistulas between the gall bladder and the intestinal tract are rare. Roth, Schroeder, and Schloth[1] were able to find only forty-three cases among 10,866 autopsies—less than 0.5 per cent.

The clinical diagnosis of internal biliary fistula is almost impossible without the aid of roentgenologic studies. Judd and Burden[2] report that in their 153 cases of spontaneous biliary fistula, the diagnosis was made only twice. Since Carman and Miller[3] first reported their case in 1917, some forty-five cases of spontaneous internal biliary fistula have been reported as diagnosed preoperatively by means of x-rays. In about half the cases there is no history of biliary symptomatology. The number of cases diagnosed roentgenologically is small when compared with the number of such cases actually found at operation or autopsy, for the diagnosis can be made only when air or barium is found to

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be present in the biliary tract. This is rare, since even the direct introduction of air or lipiodol through a T-tube in the common bile duct will outline the biliary radicles appreciably only if the pressure is raised to a high degree—very much higher than could be expected to occur on the intestinal side of a biliary fistula. Theoretically, air or barium can enter the biliary tract by reflux through the ampulla of Vater, but the number of such cases proved by operation or autopsy is small (five according to Sickels and Hudson[4]).

Spontaneous fistulas occur between the gall bladder and the duodenum in about 50 per cent of the cases, and almost always as the result of the presence of stones and infection in the gall bladder. Cases have been reported[5] where stones were seen at operation in the process of advancing through the lumen of the fistula. A large gallstone will thus get into the intestinal tract, become impacted in the small bowel, and give rise to intestinal obstruction (case 2). In about 25 per cent of the reported cases, a perforating peptic ulcer is the causative agent (Kantor and Jaffin,[6] Lonnerblad,[7] and so forth). Finally, an eroding malignancy of the stomach or gall bladder may be responsible. Stones may, however, erode from any portion of the biliary system into a neighboring organ. Gutmann,[8] for example, cites a case of a cholecystonephric fistula, the stones being passed by way of the urinary tract.

Visualization of the gall bladder in the presence of a biliary fistula is not to be expected; first, because the gall bladder is diseased, and, secondly, the dye, even if it could be concentrated, escapes immediately into the intestine through the fistula.

The two cases that follow are of special interest in that in the first instance the fistula was detected by virtue of the presence of air in the bile passages and, in the second, not only was a fistula shown by the passage of barium into the biliary tract, but in addition a gallstone was visualized in the ileum producing intestinal obstruction. Both were diagnosed correctly.

Case 1. D. G., a 66 year old Jewish housewife, entered the hospital on August 23, 1936 with a chief complaint of abdominal pain of 1 week's duration. Although the history was not reliable, it appeared that the pain was constant in the midepigastric region, and radiated occasionally to the right flank. It kept her awake the whole night preceding the day of admission. No association of the pain with meals could be established. The patient felt quite nauseated, but she could not vomit. There was no history of icterus, tarry or clay-colored stools, or chills and fever.

The physical examination on admission revealed a moderately ill patient. The temperature was 101° F., the pulse was 110, the respirations were 30. There was no evidence of jaundice. Examination of the abdomen revealed generalized tenderness over the right upper quadrant, most marked at a point near the tip of the 12th rib. There was increased resistance to palpation and dulness to percussion. The liver and spleen could not be felt and seemed normal in size by percussion. No abnormal masses were felt.

Laboratory findings revealed a leukocytosis of

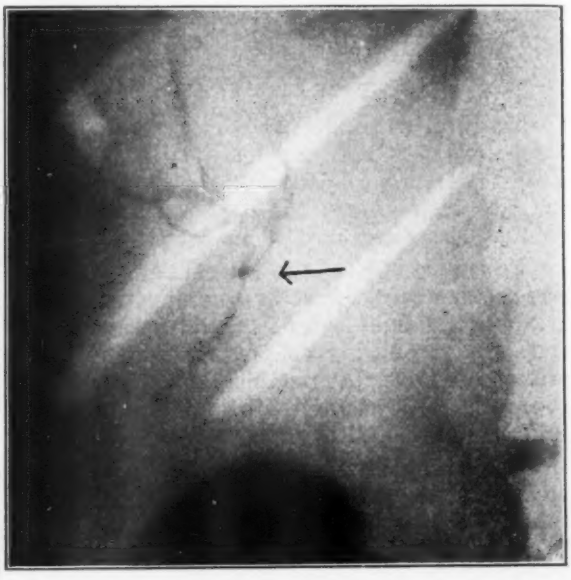

FIGURE 1.

Film of the gall bladder region showing air in the bile passages.

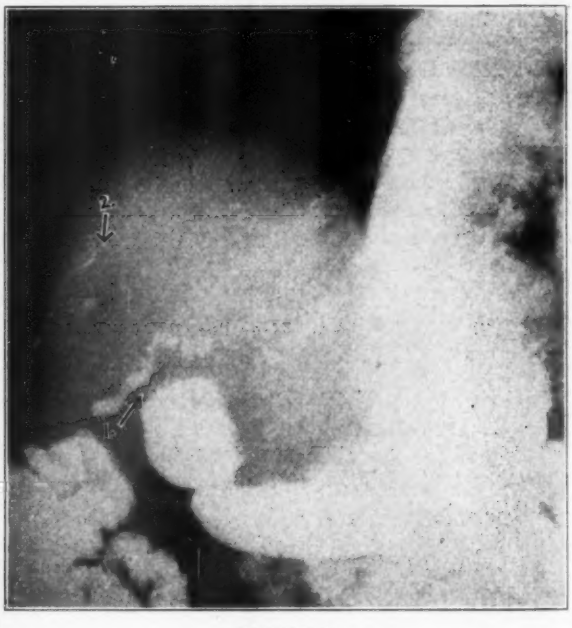

FIGURE 2.

This film shows filling of the gall bladder and some of the biliary radicles with barium. Arrow (1) indicates fistulous communication between the gall bladder and the duodenum.

18,000 with a neutrophile count of 80 per cent. Several urine examinations showed a heavy trace of albumin, 12 red blood cells and 3 white cells per high power field with many hyaline and granular casts. Stool examinations were normal. Blood chemistry

showed: nonprotein nitrogen—41, total protein—7.24, albumin—5.20, globulin—2.04, icteric index—8.75. The Hinton and Kahn reactions were reported as negative.

A scout plate of the abdomen revealed branching, linear air shadows spreading through the liver and corresponding to the biliary tree. A cholecystographic examination showed no gall bladder outline, but the areas of increased radiance just described were again present (fig. 1).

During 5 days of observation the patient's temperature fell to normal and she was symptom-free. A barium enema was given for the possible presence of a cholecystocolic fistula, but none was demonstrated. During a fluoroscopic examination of the gastrointestinal tract, barium was seen passing from the duodenum to the biliary radicles. X-ray plates confirmed this observation (figs. 2 and 3).

FIGURE 3.

More complete filling of the bile passages is here shown. The patient has been allowed to lie on the right side for 15 minutes.

Air in the biliary tree has been observed only seven times (Von Friederick,[9] Ohnell and Linbloom,[10] Graeberger,[11] Busi,[12] Alberti,[13] Beutel,[14] and Candel and Wolfson[15]).

Case 2. A 74 year old Jewish housewife, for 3 weeks before admission to the hospital, had attacks of upper abdominal pain, nausea, vomiting, and constipation requiring repeated catharsis. These symptoms were aggravated during the 3 days preceding admission.

Three years previously there had been a rather severe attack of upper abdominal pain associated with the same symptoms. The patient denied jaundice and tarry or clay-colored stools. There had been no abdominal operations.

The physical examination revealed a moderately ill patient. The abdomen was distended, but, according to the patient, was no different than usual. There was generalized abdominal tenderness at the first examination, but subsequently only in the left lower quadrant. No spasm was present and no masses could be made out. There was audible, but not hyperactive, peristalsis throughout the abdomen. The pulse was good, there was no vomiting, and the patient was passing small amounts of gas per rectum. However, because of the patient's advanced age and because the obstruction seemed to be incomplete, expectant treatment was adopted.

Laboratory findings revealed the following: Repeated urines showed a few white and red blood cells with occasional hyaline casts. The white blood cell count varied from 8,000 to 18,000. The polymorphonuclear count varied correspondingly from 74 per cent to 86 per cent. Several stool examinations showed a small amount of occult blood. Nonprotein nitrogen was 64 on admission; the icteric index was 11.

A plain abdominal film revealed distended loops of small bowel (fig. 4) suggesting intestinal obstruc-

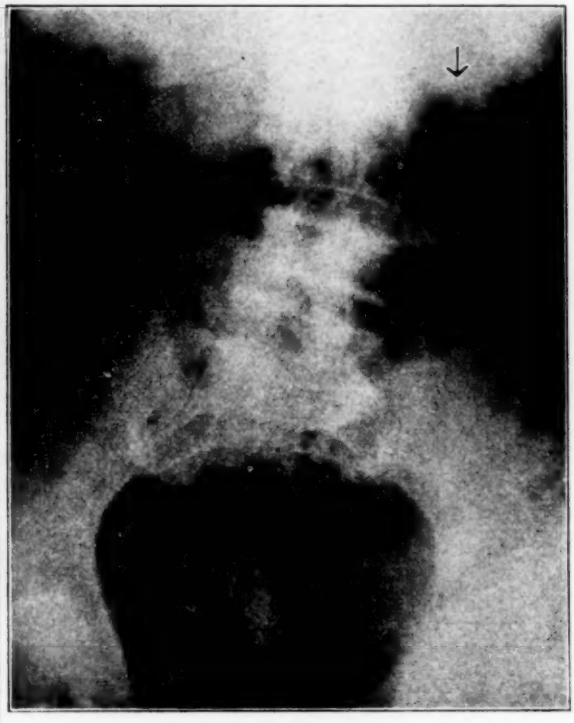

FIGURE 4.

Plain film of the abdomen showing dilated loops of small bowel.

tion. A barium enema for localization of the obstruction was unsuccessful. Following a small amount of thin barium by mouth a tab of barium was seen projecting (fig. 5) from the distal portion of the duodenal cap. The small bowel was definitely distended. A 6-hour plate revealed an ovoid area of increased radiance in the terminal ileum (fig. 6) which was interpreted as a large gallstone. The diagnosis of intestinal obstruction due to a gallstone which had gained entrance to the intestine by way of a spontaneous internal biliary fistula was made. This was confirmed at operation. A large gallstone (fig. 7) was removed from the terminal ileum about 1 foot proximal to the ileocecal valve. The small bowel was dilated and hypertrophied proximal to the site of the obstruction. The right upper quadrant revealed numerous firm adhesions which were not

FIGURE 5.

Arrows indicate fistulous tract between the gall bladder and the duodenum. Note the small, contracted gall bladder filled with barium. Loops of dilated small bowel can be seen in the right lower half of the film.

FIGURE 6.

Arrow points to ovoid shadow which was interpreted to be an enterolith (gallstone).

Note the "herringbone" appearance of the loops of dilated small bowel proximal to the site of the obstruction.

disturbed. An enterostomy was done about 6 inches above the site of the obstruction.

The convalescence was essentially uneventful until the day before the patient was to be discharged when she suddenly became cyanotic, lapsed into coma and died. Autopsy showed a massive pulmonary embolus. Just beyond the pylorus in the first portion of the duodenum a sinus tract was found running to a small, contracted gall bladder forming a spontaneous cholecystduodenostomy. Numerous periduodenal adhesions were present.

FIGURE 7.

The gallstone removed at operation.

SUMMARY

1. A brief consideration of the main features of spontaneous internal biliary fistulas is presented.

2. Two cases of spontaneous internal biliary fistula, correctly diagnosed roentgenologically, are described.

We express our gratitude to Dr. Walter S. Levenson for permission to include Case 2 in this report.

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OXYGEN IN THE TREATMENT OF CORONARY OCCLUSION IN THE HOME

BY ALEX. M. BURGESS, M.D.,* AND FRANCIS H. CHAFEE, M.D.*

OXYGEN therapy has taken its place as an indispensable factor in the treatment of many forms of pulmonary and cardiac disease. Its importance in coronary occlusion, although emphasized in a number of recent papers,[1, 2, 3] has not been sufficiently recognized. This is evidenced by the fact that in a survey of a number of modern textbooks and systems of medicine the writers have found but one reference to the subject. In the section of his textbook on the treatment of thrombosis of the coronary arteries White[4] makes no mention of oxygen, although elsewhere, under the treatment of congestive heart failure, the following statement occurs, "Oxygen inhalation (40-50 per cent), useful for certain pulmonary conditions, has as yet little or no place in congestive failure except in giving temporary relief when there is marked Cheyne-Stokes respiration or perhaps in helping patients critically ill with coronary thrombosis." It is understood that this statement is to be revised in the forthcoming edition of the book.[5] The fact, however, that this is the only advice that the general practitioner can get on the subject from the careful study of modern textbooks is perhaps justification for this communication. In addition it is our hope to show that adequate treatment with oxygen can be carried out by the physician in the home—the place, by the way, where the majority of cases of acute coronary thrombosis are, and should be, treated.

An extensive consideration of the physiologic principles on which this form of therapy is based will not be attempted. A very clear and concise review of this subject is given in the recent article by Barach and Levy.[1] A few of the fundamental facts are, however, worth mentioning. Muscular contraction depends on the breakdown of phosphocreatin or "phosphagen" and the breakdown of glycogen to lactic acid. In moderate and severe exercise the second process predominates. If exercise of any severity is prolonged, a considerable accumulation of lactic acid takes place. To oxidize part of the lactic acid and resynthesize glycogen from the remainder, oxygen is needed, and without it exhaustion ensues and functional recovery of the muscle is impossible.

With the cardiac muscle it has been shown that even under conditions of starvation[6] or of exhausting exercise,[7] such that the glycogen of the skeletal muscles has been decreased to a minimum, the heart maintains its glycogen stores intact *so long as the oxygen supply remains undiminished.* In cases of oxygen deficiency, however, it has been amply demonstrated that the result of the anoxemia is reduction of cardiac glycogen and, therefore, reduction in the power of cardiac contraction.[8]

Thus, the importance of oxygen treatment is quite evident in every case of cardiac failure in which actual anoxemia is present. If there is failure of the pulmonary circulation with beginning edema of the lungs, we have a vicious circle established, in which the greater the anoxemia produced by this edema the more depleted is cardiac glycogen, the weaker the resultant cardiac action and the greater the tendency to further edema and resultant anoxemia. Barach and Levy[1] have also pointed out that in some patients anoxemia is not actually present but is prevented only by increased pulmonary ventilation, evidenced as dyspnea. The very effort of this dyspnea adds to the work of the heart, and that a sufficient increase in the percentage of oxygen in the inspired air both reduces the necessity for increased respiratory effort and lightens the cardiac burden is shown by a relief of both the dyspnea and the tachycardia.

In a case of coronary arterial disease we have to deal with a condition in which local anoxemia due to diminished or interrupted blood supply to part of the cardiac musculature is present. Here we may be dealing with no lack of normal oxygen saturation of the blood and, if such be the case, but little can be hoped from the relatively slight increase in oxygen tension in the blood that can be brought about by increasing the percentage in the inspired air. In the severer cases of occlusion, however, there is often sufficient cardiac embarrassment to cause very definite general anoxemia. The well-known picture of ashen pallor, dyspnea, basal pulmonary rales and hypotension is familiar. Here oxygen is sorely needed to break the vicious circle. Even though the condition may in some instances be temporary, we believe that the lives of many patients can be saved by the prompt application of oxygen therapy. In our judgment any person suffering from an acute attack which may be one of coronary occlusion, and who shows the slightest evidence of cyanosis, dyspnea or the presence of persistent basal pulmonary rales, should be given adequate and continuous oxygen therapy until the symptoms have been relieved for a long period—hours to days.

To be of value to the general practitioner

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for treatment in the home, oxygen therapy apparatus must be portable and durable. The ordinary oxygen tent is too bulky, too expensive and far too susceptible to aging and disintegration of its rubberized fabric to be a reasonable investment for the man in private practice. Occasionally, where such a tent is owned cooperatively by a number of men or is to be borrowed from a local hospital, it may be appropriate. In our experience, however, there are two methods of administering oxygen that are more useful under home conditions and, ordinarily, one or the other of these will be found adequate. These are oropharyngeal insufflation by means of the nasal catheter, as described by Wineland and Waters,[9] and administration by the open box method.[10]

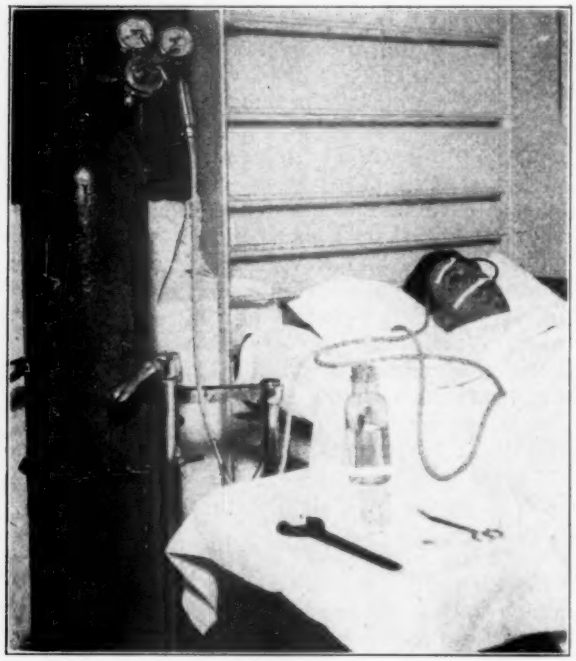

FIGURE 1.

Nasal catheter method in use. The wash-bottle should be strapped to the bed or elsewhere to prevent its tipping over. (Courtesy of the Rhode Island Hospital.)

Nasal catheter administration of oxygen has been used by us in the majority of our cases and is a very simple and useful method requiring a minimum of expensive equipment (fig. 1). For a detailed description of the technic the reader is referred to the recent article by Waters, Buerki and Hathaway,[11] in which the method in use at the Wisconsin General Hospital is described in detail. The main point to remember is that the tip of the catheter, which should have several openings, must rest in the oropharynx, about opposite the uvula. Ordinary commercial oxygen is used and is available practically everywhere. The total equipment consists of a "regulator" or reducing valve showing oxygen pressure in the tank and rate of flow in liters per minute, rubber tubing, a wash-bottle in which the oxygen bubbles through several inches of water, a glass connection between the tubing and the catheter (best made by using a medicine dropper with the tip broken off fairly well back so that there is no obstruction to free flow at this point) and, finally, the catheter. The wash-bottle should be wide, so as not to be easily tipped over, or should be strapped to the bed. The rubber stopper should not be fastened in with adhesive, as is often done, because it acts as a safety valve and blows out in case the catheter becomes loosened and the tip swallowed by the patient. Elaborate wash-bottle equipment for use in this method is commercially available but expensive. These bottles moisten the oxygen thoroughly by breaking up the bubbles, and there usually is a second bottle in each set to act as a trap for any drops of water that may be carried over from the first. The catheter should ordinarily be No. 8 to No. 12 French and should have several extra holes near the tip. Adequate oxygen concentration in the alveolar air can be achieved by this method as has been shown by the work of several investigators.[12, 13] Although the evidence as to the exact flow needed to produce a given concentration varies in the published reports, it is fairly safe to say that one should not expect clinical results with a flow less than 4 liters per minute and that usually it is best to use 6 or even 8 or higher, if the need is great and the patient tolerates it.

The open box method of administering oxygen[10] we have used less often than the nasal catheter in cases of coronary occlusion, although, in some instances, we have found it much the more satisfactory of the two. Its efficiency bears an inverse ratio to the size of the patient, which means that in the case of a large adult a relatively rapid flow of oxygen must be used. In an adult weighing 140 pounds it usually requires a flow of 4½ to 6 liters per minute to cause the patient to inhale 45 to 60 per cent oxygen, which is considerably more efficient than the nasal catheter method. If, however, the patient is restless or orthopneic, the results are much less satisfactory. In coronary occlusion many of the patients can lie flat or nearly flat and are not restless. In these the open box is most valuable and has, in our experience, occasionally given spectacular results. The apparatus for oxygen therapy by this method is now available commercially and, although it can be built fairly easily as originally described,[10] the commercial boxes, which are lighter and are equipped with interchangeable rubber hoods, will be found more convenient and probably somewhat more efficient than the homemade article (fig. 2). The concentration

of oxygen is maintained by the fact that, in spite of free diffusion upward, an adequate inflow into the bottom of the box keeps a high concentration about the patient's head.*

Oxygen therapy has been used for the last four years on all cardiac cases with definite evidence of anoxemia on the medical services at the Rhode Island Hospital. For the past two years it has been routine treatment in every patient with coronary occlusion that has shown cyanosis, dyspnea or basal pulmonary rales. During this time twelve cases have received oxygen therapy. If we add to these eleven patients seen in private practice, we have a total of twenty-three cases so treated.

As these were patients in whom cyanosis and

FIGURE 2.

Oxygen box in use. In this case the patient is an infant, therefore the thorax and arms are in the box instead of the head only, as in case of an adult. (Courtesy of the Rhode Island Hospital.)

apparent anoxemia were present, it is needless to say that they represented instances in which the condition was evidently much more desperate than those in which oxygen did not appear indicated and was not used. Eight of the twenty-three survived, and among almost all of those who died some degree of temporary improvement, such as decrease in cyanosis, dyspnea, pain or pulse rate, was noted following the institution of oxygen therapy. We gained the definite impression that among those who survived some certainly could not have done so without oxygen.

The most striking case of improvement under oxygen has already been reported in a previous paper.[10] Another example that should be mentioned was that of a 64 year old woman, who on December 29, 1934, was taken with oppressive distress across the upper chest. When seen about a half hour later she showed a fall in blood pressure, an ashen pallor, rales over both lower lobes of the lungs and a pericardial friction rub. A diagnosis of acute coronary occlusion was made and upheld by further observation and electrocardiographic studies. This patient was somewhat relieved by morphia and was placed in an oxygen box within an hour of the onset. She became quite comfortable and remained so for three days. On the afternoon of the third day the box was removed and, within a few minutes, she complained of chest oppression and difficult breathing so that oxygen therapy had to be resumed. She was kept in the box for two more days, when, after a cautious decrease in the speed of oxygen flow, it was removed without a recurrence of symptoms, and she went on to uneventful recovery. At the present time, August, 1936, she has had no return of symptoms. In the case of this woman, who weighed approximately 145 lbs., an oxygen inflow of 5½ liters per minute gave a concentration of 58 per cent, whereas a 6 liters' flow gave 64 to 69 per cent taken at the bridge of the patient's nose as she lay on her back.

*This concentration is not because of the fact that oxygen is slightly heavier than air as was inferred in the original description of the apparatus, but simply represents a balance between inflow and diffusion.

SUMMARY

Attention has been called to the lack of emphasis on oxygen therapy in the treatment of coronary occlusion and to theoretical considerations leading to the conclusion that such treatment is essential in many of these cases. Two simple and well-known methods for administration of oxygen, which are suitable for use in the home, have been mentioned and briefly described, and reference has been made to the authors' experience with the use of these methods.

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TRANSIENT HEMIPLEGIA ACCOMPANYING LOBAR PNEUMONIA

Report of a Case

BY BENJAMIN ZOLOV, M.D.*

INVOLVEMENT of organs other than the lungs is often found complicating pneumonia. The occurrence of some of these complications, such as empyema, lung abscess and pericarditis, can be easily explained by the direct spread of the inflammatory process through neighboring structures. The pathogenesis of infection in such remote organs as the brain, meninges and joints, however, has always been a moot point.

Probably the least common complication of pneumonia is the occlusion of blood vessels caused by embolism and thrombosis. Although these lesions have been recognized for many years, comparatively few investigators have had the opportunity to observe such rare clinical entities.

Mead and Bacal have reported a series of 32 cases of vascular occlusion limited to the arteries alone as a complication of pneumonias that were mostly lobar in type. In this group, the extremities were involved in 27 cases, resulting in gangrene with amputation performed as a life-saving measure. Two of the remaining cases were of hemiplegia, one of which terminated in death, the other in complete recovery. They believe that the arterial lesions were caused either by an embolism or by actual arteritis with thrombus formation. Emboli of cardiac origin sometimes have been mentioned as the source of the condition. Some authors believe that the lesions are caused by emboli arising from thrombi in the pulmonary veins. Quite often arterial occlusion is accompanied by such severe sudden symptoms that embolism is suggested rather than thrombosis.

Deshmukh describes a case of lobar pneumonia that came to autopsy showing hemorrhagic areas in the left cortical region associated with thrombosis of the smaller blood vessels in this area. He believes that these lesions are caused by the pneumococcal toxin, which has a marked injurious effect on the vascular endothelium of the brain.

Withington, in 1913, reviewing a series of over 7,600 cases of pneumonia at the Boston City Hospital, described 4 with thrombosis, 2 with emboli, and 2 with hemiplegia that went on to complete recovery. He also described 2 cases of hemiplegia following lobar pneumonia that showed no brain pathology when examined post-mortem. The scarcity of patients with pneumonic hemiplegia coming to autopsy has been the main difficulty in attempting to attribute the cause to any one or several agents. Lesieur and Froment state, "There is no doubt that a certain number of pneumonic hemiplegias are due to an acute pneumococcic encephalitis. It is equally certain that this must not be considered as the only pathogenesis to the exclusion of any other."

In a thorough study of 20,753 cases of pneumonia at the Boston City Hospital from 1915 through 1935, 10,739 of which were lobar in type and the remainder of which were classified as bronchopneumonia, only 3 cases of hemiplegia were found that could be considered definite. This group includes 2,302 cases of pneumonia that were examined post mortem, only one of which presented any pathology that was worthy of mention (case 3).

Case 1 was an 18 year old, white, single, male, who developed a right hemiplegia and aphasia 8 days after the onset of a bronchopneumonia that involved both lungs. The sputum contained *Streptococcus hemolyticus* and *B. influenzae*. The lumbar puncture was negative, as well as the Wassermann test. Almost complete recovery took place within 6 weeks. Case 2, a 32 year old, white, single, male, developed a right hemiplegia and aphasia on the third day following an attack of bronchopneumonia. Recovery here was nearly complete within 7 weeks of the onset of the condition. Case 3, a 42 year old, white, married, female, developed bronchopneumonia following an attack of measles. On the third day of the pneumonia she suddenly became unconscious and developed an aphasia followed by a mild hemiplegia on the right. Within 24 hours her reflexes were normal, though the aphasia persisted. The findings at lumbar puncture were within normal limits; the Wassermann was negative. Seven days after the first attack the patient suddenly became unconscious and died within a few minutes. At autopsy the brain was described as grossly negative, except for slight edema. Microscopic examination of the cerebrum revealed a slightly thickened piarachnoid, while scattered areas in the cortex showed a perivascular infiltration of pigmented endothelial leukocytes. The other findings were essentially normal.

The phenomenon of pneumonic hemiplegia without an "apparent" gross lesion is extremely rare. It appears usually about the third or fourth day from the onset of the pneumonia, although this period may vary a great deal. In one of Aufrecht's cases it preceded the pneumonia by 3 days, raising the question as to

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whether there was a central type of pneumonia. The complication may follow a lobar process in the lung. A higher percentage of such complications has been seen in cases showing one or more lobar involvements on the right side. Before the aphasia or paralysis there may be headache, syncope, bewilderment or vertigo. There may be paresthesia of the side followed by a fresh hemiplegia. The aphasia may come on suddenly without impairment of consciousness. Quite often, in young individuals, the sudden change brings on a fright accompanied by continued crying. The type of aphasia is usually ataxic, as in lesions of the left frontal convolutions. The patient can pronounce only a few monosyllables more or less appropriate to what he wishes to say. At first the intellect may be blunted, and he may not be able to understand what is said, but after a few hours he may indicate by gesture that he wishes to talk. In these cases there is always paralysis of the right lower facial area with the mouth drawn to the left while the tongue deviates to the right. The hemiplegia may be complete or may be limited to the tongue, face or upper extremity. The abdominal reflex is one of the first to disappear and the last to return. The tendon reflexes are sometimes affected. Quite often, the patient experiences great difficulty in swallowing. The diagnosis is sometimes difficult to differentiate from meningitis or, more rarely, brain abscess. Lumbar puncture proves one of the greatest aids in diagnosis.

The following case is almost classical in that it exhibits many of the above phenomena.

REPORT OF CASE

A 14 year old, white, American boy was admitted to the First Medical Service at the Boston City Hospital, on January 11, 1936, complaining of a head cold of 3 weeks' duration. Three days before entry he developed a dry cough and sense of tightness over the upper part of his chest anteriorly. He felt feverish at this time and went to bed. Two days before entry he began to vomit and complained of pain, aggravated by deep breathing, in the right chest. He perspired freely. The cough grew worse and was productive of thick white phlegm. No chills or hemoptysis had been experienced. His past and his family histories were essentially negative.

Physical examination revealed a well-developed and nourished boy, propped up in bed, quite dyspneic and looking extremely ill. He coughed frequently and complained of severe pain over the sternum and right chest anteriorly. The face was flushed, and the skin was hot and dry. He was rational and very cooperative. The tongue was slightly coated and grayish white, and the pharynx slightly injected. The tonsils were not enlarged. The eyes, ears and nose were essentially negative. Examination of the chest revealed diminished expansion on the right side. Impaired resonance was present over the right lower chest posteriorly, extending into the right axilla. A few, fine, moist, inspiratory rales were heard over the above area and the breath sounds were diminished. The left lung was clear and resonant. The heart showed no enlargement and the sounds were of good quality, with regular rate and rhythm. No murmurs were heard. The blood pressure was 105 systolic, 60 diastolic. The abdomen, extremities and reflexes were all within normal limits.

The temperature was 101° F., the pulse 130 and the respirations 35.

Examination of the blood showed red blood cells 4,600,000, white blood cells 14,500, hemoglobin 70 per cent, polymorphonuclears 85 per cent, lymphocytes 10, monocytes 3 and eosinophiles 2. There were no change in size and shape and no achromia, with platelets abundant. The urine was essentially negative. *Streptococcus hemolyticus* was cultured from the sputum. Blood cultures were consistently negative. An x-ray of the chest revealed lobar pneumonia of the middle and lower right lobes.

The patient was placed on routine pneumonia treatment. Two days after admission his temperature dropped to 100° F., pulse to 100, and respirations to 22. At this time there were 13,200 white blood cells. The patient felt very much better, did not complain of any chest pain, and his cough was somewhat loosened. He looked brighter. Early the same evening he complained of a dull headache limited to the left side of the head. The next morning the patient was sitting up in bed reading a newspaper. He felt quite comfortable, except for the unilateral headache. He was able to talk freely and had no special complaint. At 11:15, the same morning, a nurse discovered the patient crying and unable to talk. He seemed to understand what was said, but appeared quite frightened over the sudden loss of speech. Examination at this time revealed weakness of the right side of the face, and marked weakness of the right arm and leg. There were several irregular twitchings of the upper part of the left side of the face. The pupils were equal and regular, reacting to light and accommodation. The right knee jerk was more active and more pronounced than the left. There was a Babinski sign on the right. Cremasteric and abdominal reflexes were absent on the right side. The boy was unable to swallow any fluids, regurgitating everything through his nose. He was seen by the neurologic service shortly after this episode, and the aphasia, right facial weakness, right hemiplegia, absent abdominal reflexes and right Babinski sign were all confirmed. The diagnosis of thrombosis of the left middle cerebral artery, probably due to a toxic infectious process, was made at this time. The lumbar puncture findings were within normal limits 1 hour after the above episode.

On the fourth day after admission the temperature dropped to 98.6° F., the pulse was 100, and the respirations were 20. Physical signs in the chest showed very little change from those on entrance, though no rales were heard at this time. The patient was able to speak a few words with effort. The weakness of the right face, arm and leg was still present, although it was less than on the preceding day. He had been able to swallow small amounts of water since the acute episode. The Babinski sign was still positive with absent abdominal reflexes on the right side. On the fifth hospital day, the Babinski sign disappeared, and almost full strength returned to the right arm and leg. His speech showed marked improvement, although he stuttered a great deal when he talked rapidly. He was able to swallow water freely. The abdominal reflexes on the right side were still absent, but the cremasteric reflex was present. The facial weakness on the right side persisted. The patient continued to show gradual improvement daily. At the end of 2 weeks he was up and about the ward. The reflexes were all normal 10 days after the acute episode. The chest signs had entirely cleared. The patient had a residual

right facial weakness, which was only slightly noticeable. His speech was normal except for the occasional stuttering that took place when he talked too rapidly or when he became excited.

Approximately 6 weeks after the onset of the complication he had completely recovered from the speech defect and residual weakness.

As has been stated, the scarcity of autopsy material in these cases leads one to theorize as to the mechanism of the entire condition. Certainly all the findings cannot be explained on the basis of an acute toxic encephalitis alone. It does not seem too farfetched to picture a slowed cerebral circulation acting on a toxically injured endothelium to produce a clumping of platelets, enmeshing blood cells with a thrombus formation. Negative blood cultures appear to rule out the presence of bacteremia as the cause of the focal cerebral lesion. The likelihood of a thrombus having occurred in the above cases is certainly within reason. The extent of the lesion and the ability and rapidity of recanalization of the affected blood vessels would determine the clinical signs and symptoms. The prognosis appears good in proportion to the certainty of excluding the group of hemiplegias that are caused by brain abscess, cerebral hemorrhage, meningitis, and so forth, although it must be remembered that the progress of the pneumonia, itself, is always to be considered. It appears evident, in the light of this case and of the few cases that have been reviewed, that if the cerebral accident does not add to the gravity of the pneumonia, there is some justification in giving a rather favorable prognosis, especially when the condition occurs in young and strong patients.

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TUBERCULOSIS ABSTRACTS

Carbon Dust and Tuberculosis

Tuberculosis is comparatively uncommon in workers exposed to dusts of carbon, lime and marble while those who work in an atmosphere laden with silica particles are particularly susceptible to tuberculous infection. Coal dust is the only one to which is attributed the property of inhibiting the development of tuberculosis. Attention was called to the high resistance of coal miners to tuberculosis as early as 1862 and since then evidence has been presented that carbon particles in the lung do exert a protective influence. During recent years, however, several investigators have reported that coal miners die from tuberculosis with greater frequency than the average of the total population. And in a group of 100 old and retired coal miners in Wales, tubercle bacilli were found in the sputum of 6 on the first examination.

The type of tuberculosis that complicates anthracosilicosis differs materially from that which is usually seen. It does not extend progressively downward from the apical or subapical region, but is found scattered throughout the lungs among the silicotic nodules. The tuberculous lesion is usually of slow evolution, is always present in both lungs and consists mainly of caseous nodules varying in size from that of a split-pea to a walnut. Tendency toward cavitation is great, demonstrable cavities being found in 43 per cent of the patients. The excavations may attain tremendous size, sometimes occupying almost an entire lobe. They are often multiple and may occur anywhere in the lungs, but the larger ones are usually found in the upper lobes. Caseous pneumonia occurs as a terminal event in many instances. Extensive plastic changes in the pleura are nearly always associated. Effusion occurs infrequently.

The clinical picture of anthracosilicosis coexisting with tuberculosis may be either nontoxic or toxic. In the former type the patients present the usual symptoms of pneumonoconiosis, dyspnea, cough, expectoration and chest pains with slight fever in a few instances. Impairment of general health is slight. These cases are usually considered simple anthracosilicosis until attention is called to the tuberculosis by the discovery of tubercle bacilli or by roentgen-ray evidence.

The second or toxic form is characterized by extensive cavitation, the clinical picture being that of rapidly progressing tuberculosis. In addition to the local symptoms, there is always severe constitutional disturbance, as evidenced by fever, loss of weight, loss of appetite, weakness and profuse sweating. Fever is a predominant feature of this form. It is present throughout the entire course of the disease and is septic in type, the afternoon temperature reaching 103° or 104°F. in the majority of patients. Repeated and profuse hemoptyses occur in many instances.

It is difficult to determine the frequency with which tuberculosis coexists with anthracosilicosis in coal miners because data from death records are concerned chiefly with individuals treated at home and the detection of tuberculosis occurring coincidently with pneumonoconiosis may require prolonged observation, repeated analyses of the sputum and serial roentgenographic study.

Anthracosilicosis and Tuberculosis, Martin J. Sokoloff, Am. Rev. of Tuberc., Nov., 1936.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 23051

PRESENTATION OF CASE

A 62 year old American engineer was admitted complaining of a mass in the abdomen and cough.

The patient had been a known syphilitic for 36 years and had been treated with mercury inunction and intravenous arsphenamine. His last course of treatment had been completed 15 years ago, although since that time he had received tryparsamid three or four times a year. Serologic tests were said to have been negative for the past 13 years. About 8 weeks before entry he developed a dry hacking cough, which appeared after a minor injury to his abdomen. The cough persisted until 5 weeks before entry, when it became paroxysmal in character and productive of thin, watery, mucoid material. Examination by a physician at that time showed a mass the size of a small grapefruit in the midepigastrium and considerable abdominal distention. The patient was sent to a hospital where he remained for 2 weeks. While in the hospital he developed anorexia and began to lose weight. He was informed that the mass was within his liver, but studies of his gastrointestinal tract were said to be negative. Three weeks before coming to this hospital he again consulted the physician who had treated him previously for syphilis; this time because of increasing size of the mass and progressive distention. He was advised to enter still another hospital, which he did. There he was given 50 to 75 drops of potassium iodide three times daily. Under this regime he rapidly lost strength, became markedly distended and short of breath, and the abdominal mass continued to increase in size. There was no associated nausea, vomiting, hemoptysis, hematemesis or melena. For about 3 months his bowel movements were slightly costive. Three days after his discharge he entered this hospital.

Six years before entry the patient had a prostatectomy performed, and 2 years later a bilateral inguinal hernia repair was done. In the interval between the two operations the patient had jaundice, the details of which were not noted.

Physical examination showed a well-developed, slightly undernourished man lying in bed with obvious respiratory difficulty. The pupils were small, reacted to accommodation, but exhibited very sluggish response to light. The breath sounds at the right base were diminished in intensity, and there was impaired resonance in this region. The remainder of the chest was clear. The apex beat of the heart was in the midclavicular line in the fourth interspace. The sounds were of poor quality and obscured by the rather coarse breath sounds. The blood pressure was not recorded. The abdomen was markedly distended and tense, with bulging of the flanks. There were tympany in the region of the umbilicus and slight shifting dulness in the flanks. In the midepigastrium, extending halfway to the umbilicus, was a stony hard, lobulated mass. Rectal examination was noncontributory. The remainder of the examination was negative.

The temperature was 99.6° F., the pulse 105. The respirations were 24.

Examinaton of the urine showed a specific gravity of 1.020 with a slight trace of albumin. The sediment contained 18 to 20 white blood cells but was otherwise negative. The blood showed a red cell count of 4,400,000, with a hemoglobin of 80 per cent. The white cell count was 11,000, 76 per cent polymorphonuclears. A stool examination was negative. A tuberculin test was negative. A Hinton test was negative. The nonprotein nitrogen of the blood was 39 milligrams. The serum protein was 5.1 grams per cent. The chlorides were equivalent to 99 cubic centimeters N/10 sodium chloride. Gastric analysis showed a free acid of 10 and a total acid of 32. The guaiac test gave a 1+ reaction.

An x-ray film of the chest showed mottled dulness in both lung fields, less pronounced at the apices. The mottling consisted of foci ranging from a few millimeters to a half centimeter in diameter. They were hazy in outline and confluent, particularly in the right middle lung field. The costophrenic angles were clear. The heart was within normal limits. The aorta was tortuous. A gastrointestinal series showed evidence of extensive varices within the esophagus. The stomach was displaced to the left by a soft tissue mass, the center of which lay to the left of the spine. This was said to correspond to the left lobe of the liver. The right lobe of the liver was apparently not enlarged and, if anything, was thought to be smaller than usual. The posterior portion of the duodenum was unusually long and reached high into the right hypochondrium, where there were an unusual number of gas-filled loops of small and large intestines.

The patient's condition became progressively worse, although his temperature after entry remained below 99°F. He began to vomit every-

thing ingested. Despite the fact that the abdomen was tapped and 32 ounces of fluid obtained, the distention returned very rapidly. No comment was made relative to the gross appearance of the fluid, but its specific gravity was 1.010 and the cell count showed 50,000 red blood cells and 250 white blood cells. The differential smear showed 9 polymorphonuclears, 75 lymphocytes and 16 monocytes. The respirations became progressively more difficult, and numerous bubbling rales were heard in the chest. He died on the fourth hospital day.

Differential Diagnosis

Dr. Charles L. Short: To recapitulate the history, this man of 62 contracted syphilis at the age of 26. He had had treatment consisting of arsenic and mercury for upwards of 20 years, and in the last 15 years had received tryparsamid, although the serologic tests were negative during that time. I think we have to assume he had syphilis whether or not the signs of it are found at autopsy and, since he received tryparsamid, he presumably had syphilis of the central nervous system. Six years ago he had a prostatectomy performed. This was probably for benign hypertrophy, but we are not told the nature of pathology in the gland. About 5 years before admission he had jaundice. It would be interesting to know the severity and course of the jaundice, whether it was accompanied by pain, and so forth, since I believe it was of importance, and I shall refer to it later. The present illness really started 3 months before admission, when he became slightly constipated. For 2 months he was troubled by cough, and an epigastric mass was known to be present for 5 weeks, along with the development of ascites. He lost weight and appetite during this time, and at one point in his illness he received more or less of a therapeutic test for syphilis with potassium iodide.

Physical examination confirms the presence of syphilis of the central nervous system by the pupillary findings. He had dulness and diminished breath sounds at the right base. The heart was pushed up, perhaps by fluid in the abdomen, and a stony hard, lobulated midepigastric mass was felt. The ascites was confirmed by a tap of the abdomen.

The laboratory work does not seem to be helpful in the way of differential diagnosis. He had a very rapid downhill course. The whole illness really lasted less than 2 months.

X-ray Interpretation

Dr. Richard Schatzki: The examination of the chest showed mottled small lesions scattered throughout both lung fields—less pronounced in the apices, more pronounced in the bases of the lungs. In some areas the distribution seemed to follow the course of the lymphatics. The areas were round, and most of them were not too well defined. The stomach was displaced to the left side by a shadow which corresponded to the palpable mass. The inside of the stomach showed no evidence of pathology. The small intestine, as well as the duodenum, was high in the right hypochondrium, and, therefore, the right lobe of the liver certainly could not be enlarged. If anything, it was smaller than usual. Much to our surprise, the esophagus showed extensive varicosities.

Dr. Short: Have you any opinion as to whether the mass was in the liver?

Dr. Schatzki: We thought it was in the left lobe of the liver.

Differential Diagnosis Continued

Dr. Short: He has extensive infiltration in his lungs, the nature of which is not definite just from looking at the film; and he has this mass probably in his liver, and extrinsic to the stomach. Also, he has extensive varices of the esophagus.

In the differential diagnosis of this case I can repeat that the patient had syphilis, but what evidence of it we shall find at autopsy, if any, is not apparent from the record. I believe also that he had intrinsic liver disease. He had a history of one definite attack of jaundice. He had ample opportunity for damage to his liver from antiluetic treatment and from syphilis itself, as well as, possibly, from a hepatitis of unknown but independent origin. The presence of ascites is confirmatory of this, and the demonstration of varices by x-ray I believe clinches the diagnosis of some type of cirrhosis with portal obstruction. The most likely type in view of the man's past history, it seems to me, is what is called, variously, nodular cirrhosis, toxic cirrhosis or subacute yellow atrophy. We know such cases can exist, having widespread liver destruction and regeneration, with a history of only mild attacks of catarrhal jaundice in the past, or even with no history pointing to liver disease. Could this one diagnosis explain the whole picture? Could the epigastric mass consist of large nodules of regenerated liver tissue? I believe it could, although death without jaundice and in the absence of severe hemorrhage would be unlikely from cirrhosis alone. The only out, of course, is the presence of pulmonary lesions and symptoms. In order to link up the pulmonary and the abdominal findings, unless we try to explain them on separate bases, I think we must assume that the palpable mass represents malignancy, and that assumption certainly is borne out by the character of the mass and the rapid downhill course that this patient followed. It seems unlikely that syphilis alone could explain the entire picture and the patient's death. Certainly the attempt at a ther-

apeutic test with potassium iodide does not carry much weight one way or the other.

Let us consider some of the sources of malignancy, if we assume there is malignancy in the left lobe of the liver and the lungs. Any patient who has had a prostatectomy is, of course, always under suspicion. The rectal examination was said to be negative and, in the absence of skeletal symptoms or local recurrence, extensive liver and pulmonary involvement would be most unlikely. I would, therefore, be willing to rule out the prostate as the primary source. The stomach is eliminated by x-rays here and in another hospital. We cannot say as much for the large bowel, although the negative stools and the absence of more gastrointestinal symptoms would seem to be against this diagnosis. Primary carcinoma of the gall bladder is possible. The mass seems too far to the left to be the gall bladder itself. He has no past history of gall bladder disease, unless we include the one attack of jaundice, and we would expect more pain. There are other possible sources of primary malignancy, of course, but there is no positive evidence in their favor. I shall just mention carcinoma of the bronchus or a tumor of the kidney.

Could he have extensive pulmonary tuberculosis, with the findings in the liver independent? The negative tuberculin test, of course, does not rule it out. We would like to have repeated examinations of the sputum for tubercle bacilli, which are not mentioned. On the whole, there is no evidence in favor of this diagnosis.

Finally we come to a primary growth of the liver itself. We are told that this condition arises practically always on a previously cirrhotic liver. It may be limited to one lobe. It runs a rapid, fulminating, downhill course and may metastasize, in certain cases, to the lungs. A liver with extensive areas of regeneration in a toxic cirrhosis might well favor a malignant regrowth. The two main reasons for making this my first diagnosis are, first, the absence of a primary source of malignancy and, secondly, the evidence of proper soil for such a type of malignancy, namely, cirrhosis. I should put as my first diagnoses, then, primary malignancy of the liver implanted on a toxic cirrhosis with esophageal varices, and syphilis.

Clinical Discussion

Dr. Tracy B. Mallory: Dr. Short has taken what a year or two ago would have been a long chance in making a clinical diagnosis of primary cancer of the liver. The one piece of evidence that was most essential to the chain of reasoning was one which 2 or 3 years ago we would not have known how to determine. Thanks to Dr. Schatzki the esophageal varices were beautifully demonstrated in this case. Dr. Schatzki made a diagnosis of primary hepatoma on this patient while he had him in the fluoroscopy room, I believe. Will you explain your reasoning, Dr. Schatzki?

Dr. Schatzki: This man had a palpable mass. The roentgen examination showed the mass to be extrinsic to the stomach, within the left lobe of the liver or close to it. The metastases that were visible in the chest confirmed the impression that the mass represented a malignant tumor. The demonstration of varices within the lower two thirds of the esophagus proved that the patient had an obstruction somewhere in his portal system, presumably a cirrhosis of the liver. This assumption was supported by the fact that the right lobe of the liver appeared to be small. The only logical explanation for all these findings was a cancer of the left lobe of the liver with lung metastases, the cancer developing on top of a cirrhosis of the liver.

Clinical Diagnoses

Primary carcinoma of the liver with metastases to the lung.
Cirrhosis of the liver.
Esophageal varices.

Dr. Charles S. Short's Diagnoses

Primary malignancy of the liver with pulmonary metastases.
Toxic cirrhosis of the liver.
Esophageal varices.

Anatomic Diagnoses

Toxic cirrhosis of the liver.
Primary liver cell carcinoma (hepatoma) with metastases to the lungs and the retroperitoneal lymph nodes.
Esophageal varices.
Portal thrombosis.

Pathological Discussion

Dr. Mallory: This is the first case of primary cancer of the liver that on adequate evidence has been correctly diagnosed before autopsy in this hospital, except for one or two cases where exploration and biopsy were done. The liver was markedly cirrhotic. Here is the rather small, atrophied right lobe. The gall bladder marks the division, and you can see that the major part of the specimen consists of the left lobe, which is much more coarsely nodular than the right. The nodules are different in character; whereas the nodules in the right lobe are yellow granules of liver tissue, the nodules over here in the left lobe are white, much harder and obviously neoplastic. So we have the proper gross appearance for a diagnosis of hepatoma. There is one other piece of confirmatory evidence that one could expect to see in the gross specimen. These hepatomas with great

regularity invade the hepatic veins, and it is not uncommon to see an actual tumor thrombus grow from the liver directly up to the vena cava and practically fill the right auricle of the heart with tumor. In this case it had not extended quite so far as that, but when we looked at the hepatic vein we could see a little nodule of tumor thrombus sticking out of its mouth just where it entered the vena cava. There was a large tumor mass in the region of the pancreas, and for a little while we thought there might be some difficulty in ruling out primary cancer of the pancreas; but as one dissected that further, it became obvious that the tumor lay in lymph nodes around the pancreas, surrounding it almost completely, and that the pancreas itself was perfectly negative.

The final proof of the diagnosis is, of course, the histology, and the sections show large acidophilic cells with prominent cell membranes, vacuolated cytoplasm and up to 10 to 15 nuclei in a single cell. The arrangement of the anastomosing cords is characteristic of a primary hepatoma. The lungs were littered with metastases, primarily blood-borne with secondary lymphatic extension. There was one additional gross finding, which probably had something to do with the rapidly fatal course of his disease. That was a massive thrombosis of the portal vein from the level of its entrance to the liver back as far as the mouth of the splenic vein. This thrombus in the portal vein consisted simply of blood clot with beginning organization; it was not neoplastic like the one in the hepatic vein.

Dr. Schatzki, have you ever seen esophageal varices in a case of metastatic malignancy to the liver? We have never seen them at autopsy, but the examination for esophageal varices at autopsy is distinctly less accurate than fluoroscopic examination because they collapse so readily. We might easily miss small ones.

DR. SCHATZKI: I have seen one case that had very small varices in the lower end of the esophagus in a patient with cancer of the stomach. He did not have any evidence of cirrhosis, and I thought that the small varices might perhaps be produced by metastases around the portal vein. An autopsy was not performed, and I have no proof. I do not know if varices can develop on such a basis, but I do think very definitely that it would be unsafe to assume such a possibility in a given case.

CASE 23052

PRESENTATION OF CASE

First Admission. A 42 year old American electrician was first admitted complaining of diarrhea.

The patient had entered the Out Patient Department 3 months previously complaining of diarrhea of 2 months' duration. Previous to that time he had been in good health. The diarrhea manifested itself intermittently by four to seven watery movements a day. There was no nausea, vomiting, jaundice or melena. A proctoscopy at that time was negative except for a slightly reddened, boggy mucous membrane. A barium enema was given, and this passed to the ileocecal valve without delay. At the junction of the cecum and ascending colon there was spasm, and a postevacuation film showed some enlargement of the mucosal folds. Very little barium passed through the ileocecal valve. The distal 5 centimeters of the ileum could not be filled, but the segment of small bowel above this point was normal in appearance. Another barium enema 1 week later showed no evidence of abnormality in the distal ileum. Ten days before entry the patient began to vomit moderate quantities of dark brownish material. A bowel movement at this time contained similar material. There was no associated pain, and within 24 hours the vomiting ceased. Shortly afterward a gastrointestinal series exhibited an irregular filling defect with ragged edges involving the lower third of the stomach and the base of the duodenum. The defect was sharply defined above from the normal stomach, and several small pockets of barium in the involved area had the appearance of small ulcerations. During the 2 months preceding entry the patient's weight had diminished from 195 to 183 pounds.

Physical examination showed a fairly well-developed and nourished, nervous, middle-aged man. The mucous membranes were slightly pallid. The lungs were clear. The heart was normal. The blood pressure was 110/80. Abdominal examination was unsatisfactory because of voluntary spasm.

The temperature, pulse and respirations were normal.

Examination of the urine was negative. The blood showed a red cell count of 4,300,000, with a hemoglobin of 70 per cent. The white cell count was 12,000, 76 per cent polymorphonuclears. The stools showed a 1+ reaction to the guaiac test. Gastric analysis showed a free acid of 27 and a total acid of 37, 1 hour after the test meal. A guaiac test on the fasting contents was strongly positive.

X-ray examination showed findings similar to those described in the Out Patient Department.

On the sixth hospital day a laparotomy was performed and a mass was found occupying the pyloric third of the stomach. The mass was the size of an orange, hard and nodular, and from the lower curvature there extended another mass of metastatic glands about the size of a robin's egg. No nodules were felt in the liver. An anterior gastroenterostomy was done, and

a mesenteric lymph node removed. This was reported as hyperplasia. The patient responded moderately well postoperatively and was discharged 1 month after entry.

Second Admission (6 months later). The patient returned for x-ray studies. There were no additional complaints although the patient had had occasional bouts of fever; the height of the temperature was not noted. Previous to reentry he had received an intensive course of x-ray therapy to the abdomen.

Examination of the blood showed a red cell count of 4,000,000, with a hemoglobin of 50 per cent. The white cell count was 6,500, 77 per cent polymorphonuclears. Repeated stool examinations gave negative reactions to the guaiac test.

A gastrointestinal series showed a functioning gastroenterostomy. Peristaltic waves passed down the upper half of the persisting deformity and disappeared over the antrum. The duodenum was markedly deformed, as it had been previously. There was definite evidence of enlargement of the spleen characterized by displacement of the fundus of the stomach downward. The stomach emptied normally through the gastroenterostomy stoma and pylorus. In 6 hours the barium had reached the rectum. There was some evidence of fluid in the abdomen.

The patient received further x-ray therapy to the abdomen and was discharged on the nineteenth hospital day.

Third Admission (6 months later). Following discharge the patient continued to receive x-ray treatment and felt comparatively well. During the month preceding reentry, however, he developed pain in the left upper quadrant and complained of ready fatigability. Appetite was poor, and there was progressive constipation.

No physical examination was done.

The temperature, pulse and respirations were normal.

X-ray of the dorsal and lumbar spine showed no evidence of disease. A gastrointestinal series showed very little change since the last observation. The antrum of the stomach was involved in a tumor mass; the lesion extended back to the enterostomy and no barium passed through the stoma. During the fluoroscopic examination a small amount of barium passed around the duodenal loop. At the end of 6 hours only a trace of barium remained in the stomach.

The patient was given another series of x-ray treatments and was discharged on the sixth hospital day.

Final Admission (1 month later). Following the last discharge the patient frequently had generalized abdominal cramps, which occurred very shortly after the ingestion of food or the administration of cathartics. His constipation had become quite marked. His appetite was poor and, during the week preceding reentry, he had been unable to sleep, despite the administration of many sedatives. There was a loss of about 50 pounds in weight since the onset of his illness. Three weeks before reentry he had had severe pain in the lower back, which subsided spontaneously.

No physical examination was done.

Examination of the urine showed a trace of albumin, but was otherwise negative. The blood showed a red cell count of 3,800,000, with a hemoglobin of 70 per cent. The white cell count was 9,400, 88 per cent polymorphonuclears. Repeated stool examinations showed strongly positive reactions to the guaiac test during the initial few days. Subsequently, however, the stools were persistently negative.

His condition became progressively worse, and he died on the thirty-fourth hospital day, 1 year and 3 months after his initial entry.

Differential Diagnosis

Dr. Edward B. Benedict: The chief complaint of diarrhea at once calls attention to the large intestine and makes one wonder about the possibility of an ordinary gastrointestinal upset from dietary indiscretion. As we proceed with the history, however, and find that the diarrhea was of 2 months' duration we can be sure that there was more definite underlying pathology, and we think of the possibility of mucous colitis, ulcerative colitis, polyposis or carcinoma. There was, at this time, no melena, and a proctoscopy was negative except for a slightly reddened boggy mucosa, which would make ulcerative colitis unlikely and should exclude carcinoma of the rectum. The essentially negative barium enema should also exclude ulcerative colitis, carcinoma and polyposis of the large bowel. Failure to fill the distal 5 centimeters of the ileum suggests the possibility of a regional ileitis, but the defect may have been due to spasm. Apparently when the barium enema was repeated it was found to be negative.

The history now turns to the upper gastrointestinal tract with the patient vomiting moderate quantities of dark brownish material for a period of 10 days before entry. The most common causes of hematemesis are, of course, peptic ulcer and carcinoma, although chronic gastritis may result in fairly severe hematemesis. The lack of associated pain would be more in favor of carcinoma, and the gastrointestinal x-ray showing an irregular filling defect with ragged edges, involving the lower third of the stomach, is definitely in favor of tumor. Benign tumor of the stomach might be mentioned as a possibility, although it is relatively rare and would

certainly not produce a ragged filling defect of this size. When we read that the base of the duodenum was also involved, we think of the definite possibility of malignant lymphoma; for, although carcinoma may extend beyond the pylorus into the duodenum, it is not so likely to do this as is lymphoma. The loss of weight would, of course, tend to favor a diagnosis of malignant tumor.

The physical examination and the laboratory findings are not particularly helpful. There is a moderate anemia, also consistent with malignant tumor. An increase in the number of platelets in the blood smear would help somewhat in establishing a diagnosis of malignant lymphoma, but the platelet count is not reported. Gastric analysis showed that free acid was present, which is somewhat against carcinoma, although it by no means excludes it.

At operation we have positive evidence of a hard nodular lesion of the lesser curvature with glandular involvement, but with no nodules in the liver. Apparently this was too extensive for resection, and a palliative anterior gastroenterostomy was performed. The tumor mass could have been either carcinoma or lymphoma, but the report of hyperplasia of the mesenteric lymph node that was removed probably indicates early malignant lymphoma, the characteristic giant cells having not yet appeared. The occasional bouts of fever are also consistent with a diagnosis of malignant lymphoma, and the fact that the patient received intensive x-ray therapy to the abdomen with some apparent improvement, would make lymphoma a much more likely diagnosis than carcinoma. X-ray therapy is very helpful in lymphoma but of very little value in carcinoma. At this time, x-ray examination showed definite evidence of an enlarged spleen, which might be characteristic of Hodgkin's disease.

On the third admission we find the patient's general course is downhill, with pain in the left upper quadrant and ready fatigability, all of which are best explained by a diagnosis of malignant tumor. The generalized abdominal cramps complained of in the final admission are probably due to marked involvement of the mesenteric and retroperitoneal lymph nodes, and the increasing constipation may be the result of pressure or diminished intake of food. The severe pain in the lower back might be explained by a focus of the disease in the bone marrow, although this is rare, or by pressure on nerves. At this admission the high polymorphonuclear count and the low leukocyte count are consistent with malignant lymphoma. The early symptoms on the first admission, referable to the lower bowel, may be explained on the basis of glandular involvement and irritation with resultant spasm and diarrhea.

My diagnosis, then, is malignant lymphoma of the stomach and duodenum, and I believe that the pathologist will demonstrate a generalized glandular involvement with foci of disease in the spleen and, possibly, in the liver.

X-ray Interpretation

Dr. Aubrey O. Hampton: I first saw this patient about a year following exploratory operation and gastroenterostomy. At that time he had a markedly enlarged abdomen, apparently due to fluid; and there was a large irregular tumor in the epigastrium.

At the insistence of the patient's family and of the surgeon that did the operation, we began deep x-ray therapy. To my surprise the abdomen reduced in size, and the palpable mass seemed to become smaller. At about the sixth day of treatment, when 1300 r of deep x-ray therapy had been given, the spleen could be easily palpated. We then reviewed the patient's history, previous x-ray examinations and the histology of the gland removed at operation and concluded that the patient might have lymphoblastoma. Small doses of x-ray treatment were therefore continued over a period of 1 month, until 3600 r had been given. The patient steadily improved, but the palpable mass did not disappear. More treatments were given at 2 and 4 month intervals without benefit. The x-ray examinations of the stomach showed surprisingly little change during the entire period and there was never any evidence of disease in the chest or superficial lymph nodes.

Clinical Diagnosis

Lymphoblastoma of the stomach and peritoneal cavity.

Dr. Edward B. Benedict's Diagnosis

Malignant lymphoma of the stomach and duodenum.

Anatomic Diagnoses

Carcinoma of the stomach with invasion of the duodenum, the liver and the pancreas.
Gastroduodenal ulcer.
Thrombosis of the splenic vein.
Operative scar, anterior gastrojejunostomy.
Stenosis of the common bile duct.
Icterus.
Decubitus.

Pathological Discussion

Dr. Tracy B. Mallory: The postmortem examination allows us to be definite in one respect, that the neoplasm was carcinomatous rather than lymphomatous in character, but leaves many other questions raised by his clinical record unanswered.

At the time of autopsy we found a massive

tumor, which lay chiefly in the upper half of the retroperitoneal tissues in the general region of the pancreas. It replaced the entire head and body of this organ, involved all the regional lymph nodes and was inseparable from the pyloric third of the stomach and the first two portions of the duodenum. The antral portion of the stomach was also inseparable from a large mass of tumor in the left lobe of the liver. On opening the stomach a large, completely annular ulcer involving the entire antrum, the pylorus and the first portion of the duodenum was found. The edges of the ulcer were raised and definitely indurated. Its base was necrotic and extremely friable. After an attempt was made to determine the left lateral border of the tumor, a strand of fibrous tissue enclosing the splenic artery and vein was found, which was thought to represent the atrophied tail of the pancreas, a point subsequently confirmed by microscopic examination. The splenic artery coursed along this mass uninterruptedly, but the vein was completely occluded by a friable adherent thrombus. The spleen at the time of autopsy was only slightly enlarged, weighing 200 grams. No metastases of tumor were found in the lower mesenteric or retroperitoneal nodes, and there was no peritoneal involvement. Nothing was found in the large intestine to explain the early symptoms pointing to a colonic lesion.

Microscopic examination showed that the tumor was an adenocarcinoma of rather rapid growth and poor differentiation, and quite variable in its morphology in different regions. A major portion of it was necrotic, especially the bulk of the tumor in the region of the pancreas. This was more probably caused by interference with its blood supply than by the x-ray therapy. The problem that is difficult to solve is, where was the tumor primary? Did it start in the pancreas and secondarily involve the stomach and liver, or was it primary in the stomach, extending upwards into the liver and downwards and backwards into the pancreas? The histology of the tumor is of no help in settling this issue. On the basis of the autopsy findings alone, one would strongly suspect a primary cancer of the pancreas, but the surgical exploration, if the facts recorded are accurate, would seem to place the initial lesion in the stomach.

The spleen was of definite interest since it showed histologically the typical early changes, the dilatation of the sinusoids, the fibrosis of the pulp cords and the periarterial hemorrhages, that are characteristic of the spleen in so-called Banti's disease. It is surprising that a spleen of 200 grams would have been detected clinically, but perhaps it was rotated forward and toward the midline by the retroperitoneal tumor, or it may actually have been bigger at the time of the x-ray examination than it was at the time of autopsy.

I cannot quite refrain from comment on the surgical naïveté of assuming that lymphoid hyperplasia is an initial stage of lymphoma. Lymphoid hyperplasia of the regional nodes is found almost invariably with carcinoma and, in particular, in the stomach is more frequently associated with carcinoma than with either benign ulcer or lymphoma.

The patient's terminal jaundice was explained by a stricture in the common bile duct at the point of its entrance into the head of the pancreas. A probe could readily be passed through, but the pressure from the tumor mass had flattened the duct completely. Above this point the hepatic and cystic ducts were markedly dilated, and the liver showed the early changes of obstructive biliary cirrhosis.

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1937 COMMUNITY FUND

The annual drive of the Community Federation of Boston is nearly at an end, and, undoubtedly, several millions have been raised for the benefit of some ninety odd charitable organizations in Boston. The last few hundred thousands are always the most difficult to secure, and it is hoped that physicians residing within the confines of metropolitan Boston have contributed their share to this worthy cause.

Many physicians, of course, cannot afford, and are not expected, to contribute to the Community Fund. Others may selfishly, but with a *certain* amount of justification, argue that their hospital work and their office and home treatment of the indigent are more than equivalent to generous contributions. The latter group should not fail to realize that charitable institutions caring for the sick constitute but a small percentage of the organizations included in the Federation. Furthermore, hospital work and care of the impoverished are far from "charities", in the true sense of the word, for they contribute in no small way to the prestige, knowledge and skill of the physician.

Are you satisfied that you have done your share? If not, there is still time for your contribution or for an increase in the amount you have already subscribed. GIVE FOR HUMAN NEEDS!

IMPROVEMENT IN THE CANCER SITUATION

THAT the well-planned and concentrated attack on cancer is at last bearing definite fruit is shown by two significant studies. The cancer mortality for Massachusetts in 1935 dropped definitely both in males and in females. More recently, Dr. Dublin, in the *Statistical Bulletin* of the Metropolitan Life Insurance Company for last October, brings out further evidence that the trend of cancer mortality is beginning to diminish. In the white female policyholders of the Metropolitan, the standardized mortality rate has shown no significant increase in the last quarter century and, indeed, the trend has been slightly downward. The slight increase among males in the Metropolitan group can be attributed, Dr. Dublin believes, to more adequate recognition of the existence of malignant disease.

The answer to the difference between male and female trend lies in part, Dr. Dublin believes, to the fact that roughly half the cancers occurring in women are external and may be easily recognized, whereas only a fourth of the cancers in men are external and easily recognized. This point was clearly elucidated by Lombard some two years ago. This belief is further strengthened by the observation that cancer rates in readily accessible sites have tended to diminish, whereas cancer rates in inaccessible sites have tended to increase, probably due to more accurate diagnosis.

Since there are about 135,000 deaths from cancer in this country each year, and since, at present, out of initial groups of one hundred at birth, ten men and thirteen women will die from cancer, the problem is still of real magnitude and the gains now being made should stir us to further effort. Diagnosis must be accurate, therapy prompt and well-chosen, and follow-up careful, in order that we may utilize to the full our existing knowledge and still further force the retreat of an enemy that, for the first time, shows signs of wavering.

HOMESPUN EPIDEMIOLOGY

EPIDEMIC, according to our own lexicographer, Mr. Webster, defining the adjective, not the noun, means "common to, or affecting

at the same time, a large number in a community". Our esteemed contemporaries of the daily press, taking advantage of Mr. Webster's absence from the scene of active human affairs, occasionally depart from those strict philological interpretations to which he was given. Less erudite personalities, recognizing also that language is a fluid rather than a frozen medium, find their own ways of evading Mr. Webster's pronouncements.

Thus when anterior poliomyelitis, being a spectacular disease, becomes more than normally prevalent in a community, it is mentioned in common parlance and in the press as epidemic, even if the widest stretch of the imagination could not construe it as affecting many at the same time, considering the total population. When, however, acute upper respiratory infections put a quarter of our school population in their beds or on the street, and when the stocks of handkerchiefs disappear from the counters of even the largest and most versatile department stores, the official guardians of our health will not admit that we are in the grip of an epidemic.

But that is New England for you; conservative, unemotional and substantial to the last. Our sanitary watchdogs refuse to dramatize a smart siege of the grippe, even when they might be adding dozens of inspectors to the pay roll, closing schools and placing strict injunctions on the osculatory practices of the public. An influenza epidemic in Chicago becomes the "flu" in New York, and the seasonal incidence of respiratory infections in Boston and its environs.

Actually this conservatism is a trifle disappointing. If we are forced to support, by taxation, departments of public health, why not get some fun for our money? After we have spread the word about town of a neat little epidemic of intestinal "flu" in our midst, and have prepared people to enjoy it, so to speak, it is distinctly letting us down when our official spokesmen announce that respiratory infections are at their normal seasonal incidence, and there is no cause for alarm. No cause for alarm, indeed! People *want* to be alarmed. Far from cooperating with the general practitioner, it would seem as if these traitors were trying to force state medicine on us, just by undermining the faith of our public.

As a matter of fact, it's rather comforting, when Mrs. Smith calls up to say that all the Smiths have stomach-aches and just a touch of the diarrhea, and that the baby has vomited, to be able to reply, "Yes, Mrs. Smith, that's the form the epidemic's taking this year. No, it's not because you've eaten something; it's a kind of bug that's going around."

No season would be complete without its cycle of epidemics, and no department of health is going to cheat us out of them.

OBITUARY

DR. FRANCIS DENNIS DONOGHUE

THE many-sided nature of the activities of Dr. Francis D. Donoghue, who died January 4, has made it desirable that men familiar with the individual interests of his career should present the picture of the man from their special viewpoints.

THE ADVISER TO THE INDUSTRIAL ACCIDENT BOARD

The Industrial Accident Board has lost a most valuable member of its organization in the passing of its Medical Adviser, Dr. Francis D. Donoghue. As a member of the Board at its inception in 1912, I had the fortunate privilege of casting my vote for Dr. Donoghue as the Board's first medical adviser, and I have enjoyed the added pleasure of observing him live up to every expectation held of him at the time of his appointment. As a member of the Board for many years, and its present chairman, I am in a position to pay him this last tribute of esteem and affection, on behalf of the Board and its entire personnel.

When Dr. Donoghue came to the Board as Medical Adviser, the Act was new and the Board had no policy regarding the medical features of the Act. He immediately called together representatives of the various medical societies and hospitals, and, through his initiative, rules and regulations relative to medical treatment, fees and expenses were formulated to the full satisfaction of all concerned. His valuable opinion and medical advice were not bounded by the limits of this State but were freely given to the compensation authorities of other states that constantly sought this advice. His efforts in this connection did much to cement a splendid feeling of cooperation between our Board and those of other states, as well as the medical profession and hospital fraternity.

He was never a shirker and performed the small, as well as the great, tasks with equal diligence and care. His opinions were freely accepted by the Board and readily followed. His humane and liberal character were best demonstrated in his direct dealings with the injured workmen. He was sympathetic and helpful in every legitimate case; he was quick to detect the malingerer. He inspired confidence in all who came in contact with him, because they were immediately convinced of his fairness. His passing leaves us that much poorer.—*Joseph A. Parks.*

THE SURGEON

With the death of Dr. Donoghue, this country has lost a distinguished surgeon, a profound thinker and a magnetic personality. Those who knew him well understood the mutual devotion

that existed between him and his patients. A rather brusque exterior at times covered an inherently sympathetic character.

After graduation he found it difficult to obtain the training in surgery he desired. It was necessary to make opportunities for himself, which he did. His excellent knowledge of anatomy, his faculty of looking to the ultimate causes of things, his manual dexterity, which was noteworthy, and his ability to deal with patients on their own level equipped him well for his work in general surgery.

In his early years he was fortunate in becoming an assistant to the late Samuel J. Mixter, with whom he worked considerably in the latter's private practice. He never tired of expressing gratitude for the help given him by Dr. Mixter. It was not uncommon, up to the end of his very active career in general surgery, to hear him enunciate surgical truisms accredited by him to Dr. Mixter.

From December 6, 1900, when he published in the *Boston Medical and Surgical Journal* a paper concerning a successfully treated case of placenta praevia by cesarean section—which he did in a farmhouse at Long Pond, Plymouth County—up to the last year of his life, he made time for relatively frequent, worthy contributions to the literature. His effort, after a great deal of study, really resulted in the foundation of our present State Cancer Clinics, under the direction of the Department of Public Health. He excelled in his knowledge of injuries sustained as a result of industrial accidents. He was far ahead of his time in anticipating the necessity for proper knowledge and treatment of accidents sustained in the course of employment. He foresaw legislative action with reference to industrial accidents and established the Massachusetts Society of Examining Physicians for the purpose of bringing together men interested in accident prevention and treatment.

He never forgot the obstacles he encountered in his early years in surgery and worked to demolish barriers so that others might not be so handicapped.—*William E. Browne, M.D.*

The Medical Witness

The profession of the law, no less than that of medicine, has suffered a real and substantial loss in the death of Dr. Donoghue. A familiar figure in the trial courts of the Commonwealth for more than a generation, as an expert medical witness in the trial of cases involving personal injury, he won a deservedly enviable reputation. His professional qualifications for this important work were exceptional. He was excelled by no one of his time in the attributes so essential to successful medicolegal work. His profound medical knowledge, unusual mental alertness, rare sense of humor, pleasing address, simplicity and clarity of expression and, with the years, his ever-broadening experience following innumerable trial contacts, made him a most dangerous witness for the partisan examiner, and an equally serviceable one to a just cause. But beyond all this, although he properly considered it to be his duty to interpret the medical picture as favorably to the interest of his client as his inherent professional honesty would permit, he never allowed it to obscure his duty to the Court itself. He never permitted himself to become ridiculous or petty, or to indulge in exaggerations of conditions or prognoses, which are all too commonly observed in the testimony of so many medical witnesses. At all times to command the fullest confidence of the Court was his first and greatest concern. He won it and held it in amplest measure. His pride in it was justified. Through it there grew up, during the years, mutual attachments of a professional and personal character that were both wide and intimate. Many members of the Massachusetts judiciary will mourn the loss of a rare friend; all of them will be deeply sensitive of a loss common to the legal and medical professions by the end of an eminent service to both.—*(Judge) Nelson P. Brown.*

The Bibliophile

Dr. Donoghue was quietly interested in good books in all fields of knowledge. He set himself the task of gathering the works of the classical authorities on industrial diseases and amassed a collection, unparalleled in this country, of the various editions of Agricola, Biringuccio and Ramazzini. More recently he became enthralled with the beauty of the early printed book of the fifteenth century. On his annual trips to Europe he made it a point to visit booksellers of all kinds and, in addition to the books on industrial medicine, he gathered specimens of incunabula, and had amassed a small collection of this kind of book. He had plans for complete annotated bibliographies and also for papers on the classical authors of industrial medicine.

As a collector he was a great enthusiast but always retained his clear-minded, good judgment in his transactions with the various book-dealers with whom he came in contact. Scholarship and the literature of his specialty have suffered a great loss in his passing.—*James F. Ballard.*

The Citizen

Dr. Donoghue had a kind of personality that would have lifted him to eminence in any calling. He had something superior to knowledge, a sense of knowing that he knew. It was this faculty, rising to a sharp perception of values,

that made his views always colorful and impressive. His approach to public affairs ran, of course, through the avenue of his profession, but extended immeasurably beyond. He understood public finances, political problems, human life.

It was my privilege, while in the State Senate, to assist him in the passage of a resolve that established the first commission for the study of cancer. He was deeply interested in the enactment of compensation for industrial accidents, and his official connection with the Industrial Accident Board was one of the fortunate elements in the operation of the law. He represented Massachusetts at the International Cancer Congress at Brussels and twice represented the United States at international conferences on industrial accidents, once at Amsterdam and once at Geneva.

While I was mayor I frequently sought the advice of Dr. Donoghue on matters relating to public health and public safety. They embraced such topics as water supply, sanitation, hospitalization, recreation and many others touching generally upon public welfare. He was the mayor's representative without pay upon the commission on school buildings when first organized. He gave generously of his splendid talents for the public good. His physical and mental resources seemed inexhaustible.

Frank Donoghue was a fascinating companion, a wise counselor, a true friend.—*Malcolm E. Nichols.*

The Friend

We have lost a friend. Though not seen constantly, he was always there for friendly greeting or for consultation. The keen mind, the master knowledge of his profession, the genial interest in humanity, the uncanny capacity to pick the false from the true, however speciously hidden, the efficient memory, the organizing talent, were always available.

He had strong likes and dislikes. He made a host of friends and some enemies. He was brilliant in repartee, a "caustic but fair critic, dogmatic but open-minded". Woe to the superior person with limited knowledge of his subject who attempted to talk down to him. He was critical of his friends for their benefit as he was critical of himself.

Basically he was a kindly and sympathetic person. The friend who sought him in need was sure of practical suggestions and personal assistance. He embraced his friend's cause and made it his own. He was a believer in good works as well as in prayer.

His was an outstanding dynamic personality in a period in which standardization is tending to abolish personalities. His like we shall not see again. We have lost a friend and are missing him.—*Timothy Leary.*

THIS WEEK'S ISSUE

Contains articles by the following named authors:

Guthrie, Riley H. M.D. University of Tennessee, College of Medicine, 1921. Diplomate of the American Board of Psychiatry and Neurology, 1935. Formerly, Assistant to the Commissioner, Massachusetts Department of Mental Diseases. Now, Chief Executive Officer, Boston Psychopathic Hospital. Address: Boston Psychopathic Hospital, Boston, Mass.

Dayton, Neil A. M.D. Ohio State University College of Homeopathic Medicine, 1915. Director, Division of Statistics and Research, Department of Mental Diseases, State House, Boston. Director, Division of Mental Deficiency, Department of Mental Diseases, State House. Instructor in Psychiatry, Tufts College Medical School. Chairman, Committee on Nomenclature and Statistics, American Psychiatric Association. Address: Room 167, State House, Boston, Mass. Their subject is "The Incidence of Alcoholic Psychoses in Massachusetts, 1917-1935." Page 193.

Altman, William S. M.D. Tufts College Medical School, 1927. Assistant Radiologist, Beth Israel Hospital. Consultant Radiologist, Norfolk County Hospital. Instructor in Radiology, Tufts College Medical School. Address: 32 Spear Street, Quincy, Mass.

Field, Eugene A. M.D. College of Physicians and Surgeons, New York, 1935. Resident in Radiology, Beth Israel Hospital. Address: 116 Riverway, Boston, Mass. Their subject is "Spontaneous Internal Biliary Fistulas." Page 199.

Burgess, Alex. M. M.D. Harvard Medical School, 1910. F.A.C.P. Physician-in-Chief, Medical Department Charles V. Chapin Hospital, Providence. Visiting Physician, Rhode Island Hospital. Chairman of Division of University Health and Assistant Professor of Biology, Brown University. Address: 454 Angell Street, Providence, R. I.

Chafee, Francis H. M.D. Harvard Medical School, 1931. Assistant Visiting Physician, Rhode Island Hospital. Visiting Physician to the Outpatient Department, Charles V. Chapin Hospital, Providence. Address: 454 Angell Street, Providence, R. I. Their subject is "Oxygen in the Treatment of Coronary Occlusion in the Home." Page 203.

Zolov, Benjamin. M.D. Tufts College Medical School, 1934. Formerly, Intern in Pathology, Worcester State Hospital, on First Medical Service, Boston City Hospital, and in Obstetrics, Chicago Maternity Center. Address: 296 Congress Street, Portland, Me. His subject is "Transient Hemiplegia Accompanying Lobar Pneumonia. Report of a Case." Page 206.

The Massachusetts Medical Society

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Case Histories

No. 5. Septic Miscarriage

Mrs. E. F., a white woman, 28 years old, was seen in the hospital October 10, 1922. The patient had been curetted 2 days before for bleeding following miscarriage. She had had a chill previous to entry. Her temperature was 105°F., and her pulse was 130.

There was no family history of tuberculosis, diabetes or nephritis. Her father had died of pernicious anemia. Her mother was alive and well. As a child she had measles and two acute middle ears that ruptured spontaneously; her tonsils and adenoids were removed in 1913; and she always had some trouble with her right hip, which she attributed to the result of her own breech birth. On January 4, 1922, she was operated on because of sterility. The uterus was curetted, plastics were done on both ovaries, the uterus was suspended and the appendix was removed.

Catamenia had begun at 14. For the last few years it had been of the 28-day variety, lasting 3 days, with no pain.

On physical examination, she was a well-developed and nourished young woman. Her normal weight was 128 pounds. Her teeth were well cared for. The mucous membranes were of good color. Her lungs were normal, resonant throughout, and there were no rales. Her blood pressure was 124/68. Her pulse at the time of examination was 130. Her heart was rapid, and no murmurs were heard. Her abdomen was negative. The extremities were normal. No glands were palpable. No vaginal examination was made. The lochia was scanty, rather pale, with a somewhat strong odor but not foul. A diagnosis of "septic miscarriage" was made, and conservative treatment instituted.

A blood Wassermann was negative. The patient's blood was compatible with the husband's. No cultures or smears were taken. No further blood studies were done. A urine specimen showed a specific gravity of 1.020, a slightest possible trace of albumin, no sugar and a sediment containing a few white cells, no casts and no blood.

The patient's last period was June 24, 1922. On September 5, 1922, she had some dark discharge. On September 15 she flowed bright blood, passed a good-sized clot, and lost some water. On September 17 she passed a very large clot. A consultation was held, a diagnosis of inevitable miscarriage made and, because of the bleeding, it was advised that the uterus be emptied. This was done on September 20, when she was 3 months pregnant. The cervix was packed with sterile gauze for a day, and the uterus was emptied of a twin pregnancy on the day following. The patient remained in bed for 10 days, with a normal pulse and temperature.

On October 7, while still in the hospital, she began to bleed so freely that it was deemed absolutely essential to curette the uterus for retained products. A good-sized piece of placenta was obtained, but the curettage, as always practiced in those days, was done much too vigorously. The subsequent history of the case showed that the curettage probably spread an infection that was already existing, but that was well walled off.

Following examination in the hospital on October 10, Mrs. F. was placed in Fowler's position, an ice pack was applied to the fundus, 1 dram of ergot was administered three times a day for 3 days, the windows in her room were opened wide, and she was given by mouth all the food and all the liquids that she could take. Physical examination on October 11 showed no tenderness in the abdomen and no tenderness in the legs. Her temperature in the morning was 102.8° and in the afternoon 104°F. Her pulse in the morning was 120 and in the afternoon 130. There was no change in lochia. On October 12 there was no change noticed on physical examination. Her temperature in the morning was 99.8° and in the afternoon 103°F. Her pulse ranged from 100 to 120. On October 13 her temperature ranged from 99° to 101°F. and her pulse from 90 to 100. On October 14 her temperature in the morning was 98.6° and in the afternoon 101°F. Her pulse ranged from 90 to 100. On October 15 her temperature was normal both morning and afternoon, and her pulse did not go above 90. There was no tenderness in the abdomen, and the lochia had entirely ceased. Her temperature and pulse remained normal. Vaginal examination at the end of another week showed that the uterus was in good anterior position and well involuted; there was no pelvic tenderness and there were no masses in the pelvis. She was allowed up and about for 3 days and discharged relieved.

This case shows several mistakes in treatment. While the miscarriage seemed inevitable, the amount of bleeding was not enough to have made it essential that the uterus be immediately emptied. The fact that the membranes had ruptured precipitated operation, because in 1922 it was felt that a dry uterus was

*A series of selected case histories by members of the Section will be published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the Section.

very apt to lead to infection. If this patient had been conservatively treated and if the uterus had not been emptied, unless the amount of bleeding made operation essential, it is quite probable that nature would have emptied the uterus spontaneously.

The curettage was done much too vigorously and undoubtedly played a part in the subsequent infection. While bleeding may make it essential that the uterus be entered in a septic case, the uterus must be entered as carefully and as gently as possible.

No uterine douches and no specific medication were given.

Today the treatment of septic miscarriages is 100 per cent conservative. Without bleeding, the uterus is never invaded. No douches are ever given. The patient is placed upright in bed to facilitate postural drainage. An ice bag is put on the lower abdomen. Some form of ergot is given to stimulate uterine contractions. Blood culture and cultures and smears from the vagina and uterus may give a definite bacteriologic diagnosis. In the presence of hemolytic streptococci, prontylin, 10 grains every 4 hours, *may* prove to be of specific value.

MISCELLANY

SILICOSIS AND ALLIED DISORDERS

The medical committee of Air Hygiene Foundation, headed by Dr. A. J. Lanza, chairman, announces the completion of a comprehensive treatise on "Silicosis and Allied Disorders." The committee has attempted to bring together in one report a summary of those phases of pneumoconiosis which have been the subject of research, those which have been settled more or less conclusively, and those demanding further investigation.

The report, designed for industrialists as well as physicians, offers a practical, six-point program of health control for the "dusty trades". Recommendations for research along four fundamental lines are submitted to the Foundation in the report. These researches represent those which the committee agrees are most urgently needed at the present time to increase our knowledge of pulmonary diseases that may result from air pollution.

Collaborating with Dr. Lanza in writing the review were the seven members of his committee and Miss Eleanor Rantoul, research assistant, Industrial Health Section, Metropolitan Life Insurance Company. Members of the medical committee are the following: Dr. Leroy U. Gardner, Saranac Laboratory; Dr. S. R. Haythorn, Singer Memorial Laboratory; Dr. T. Lyle Haslett, Westinghouse Electric and Manufacturing Company; Dr. R. R. Jones, Bell Telephone Company; Dr. E. P. Pendergrass, University Hospital, Philadelphia; Dr. O. A. Sander, Milwaukee, consultant, and Dr. R. R. Sayers, U. S. Public Health Service.

The conclusion of the report is a summary, which follows:

The extensive studies of silicosis in this country and abroad have given us a very clear idea of the cause of this disease, its pathology, its complications and its prognosis. On the basis of our present knowledge, it may be said that:

1. Silicosis results from the inhalation of dust containing free silica.

2. The time required for the development of silicosis varies from a few years to 20 or more, depending on the concentration of silica particles in the air and the constancy of exposure.

3. Beginning silicosis is recognizable only by properly taken roentgen films of the chest; small nodular shadows distributed throughout both lungs are the characteristic appearance. There are no symptoms or physical signs associated with early simple silicosis that can be depended upon for making a diagnosis.

4. Associated with silicosis is a marked predisposition to tuberculosis.

5. Silicosis can be prevented by protecting the industrial worker from inhaling silica dust. This may be accomplished by engineering measures.

6. Concentrations to which the dust must be reduced in order to be safe have not been absolutely determined.

7. Industrial dusts, containing silica, are frequently not all silica, being mixed with other materials. It is possible that some of these nonsilica components may prevent or modify the action of silica in the body and may alter the tendency toward complicating silicosis.

8. Asbestos, which is a silicate, is the only dust other than free silica which has been shown to cause a lung fibrosis with disability.

9. Simple, that is, uncomplicated, silicosis, as seen in industries in the United States, causes relatively little severe disability.

Control of silicosis calls for adequate medical and engineering measures. A practical program of preemployment examination of all workers exposed to dust containing silica is essential. Such examinations should include a complete physical examination of each individual, a careful history of past occupational exposure to dust, and a chest roentgenogram with standard technic.

The value of such procedure with periodic reexaminations may be summarized as follows:

1. Protection of prospective employees, who may have diseased lungs, from silica exposure.

2. Discovery of the hazardous jobs, as a result of finding the silicosis cases, in order to eradicate these hazards.

3. Discovery of active and open cases of tuberculosis that are in contact with other employees, especially protecting the younger workers, who are susceptible to contact infection.

4. Discovery, through periodic reexamination of those with inactive tuberculosis, of any reactivation of their tuberculosis before they have been active

too long, so as to protect especially the younger employees. This also offers the infected individual a better chance for a cure of his tuberculosis.

5. Discovery of early cases of simple silicosis through periodic reexamination to give them added protection from further excessive dust exposure.

6. Determination, through periodic physical reexamination, of the adequacy of the dust control program.

A careful consideration of the foregoing summary as well as the varied experiences of the members of the medical committee clearly indicates that there still is a wide field for research in the matter of pulmonary dust diseases.

The following research projects are listed, therefore, for the consideration of Air Hygiene Foundation:

A. The relations between silicosis and tuberculosis, with especial reference to the question of activation or reactivation of tuberculous infection consequent upon exposure to silica, especially in massive doses, over a short period in the absence of definite evidence of silica action (nodulation) in the tissues.

B. The effect of other substances upon the action of silica in the body. It has been claimed that where other substances are present in the dust with silica, they may retard the action of silica so as to prevent, or, at least, delay, the development of silicosis. On the other hand, certain other substances have been credited with greatly intensifying the action of silica.

C. The mechanism by which silica exerts its injurious effects upon the body. The pathologist can demonstrate what silica does in the pulmonary and other tissues of the body, but so far no reasonable explanation has been offered or demonstrated as to why silica acts as it does.

D. The technic of roentgenography. In the last few years the technic of taking x-ray films has greatly improved. Inasmuch as it would appear that to an ever-increasing extent preemployment medical examinations will include chest roentgenograms, a man may be hired or rejected on the basis of his x-ray film. It is therefore evident that the very best technic that can be evolved is the least with which industry can be satisfied.

SCIENTIFIC EVENTS FOR THE YEAR

Among the notable achievements in psychology and psychiatry for 1936 listed by Science Service are the following:

Learning may be observed at the moment it takes place, indirectly through observation of the brain's electric potential during the process, Dr. H. H. Jasper, Brown University, found.

Use of the rhythmic electric impulses from the brain to locate defective areas in that organ was reported by Drs. H. H. Jasper and H. L. Andrews, Brown University.

Brain waves occur in unborn guinea-pigs at least 12 days before normal birth, Drs. Leonard Carmichael and Charles S. Bridgman, University of Rochester, and Dr. H. H. Jasper, Brown University, observed.

Identical twins have identical patterns of brain rhythms, Drs. Hallowell Davis and Pauline A. Davis, Harvard Medical School, found from the study of 18 twin pairs.

Effects of sedative and other drugs upon the brain are partially revealed by the brain's electric potentials, Dr. W. G. Lennox and Dr. and Mrs. F. A. Gibbs, Harvard Medical School, found.

The reflex leg movement ordinarily made by a rat startled by a noise can be "forgotten" if the noise is repeated, it was found by Drs. Walter S. Hunter, Brown University, and C. Ladd Prosser, Clark University, who demonstrated that spinal reflexes can be extinguished and then restored.

Ability of a person to adjust the frequency of a tone until it appears exactly half as high in pitch as another was demonstrated in experiments by Drs. S. S. Stevens and J. Volkmann, Harvard University, and Dr. E. B. Newman, Swarthmore College, who built a numerical scale of psychologic pitch in this way.

Experimental confirmation of the theory that tones of differing pitch are distinguished because they affect different areas of the ear's basilar membrane was obtained by Drs. Hallowell Davis, S. S. Stevens and Moses H. Lurie, Harvard University. Dr. Stevens also found that the human ear can act as a radio loud speaker, converting electric impulses of the radio receiver into tunes readily identified by the "listener".

Startle, such as that produced by a revolver shot, causes two distinct types of emotional response—first, an instantaneous involuntary and universal reaction, and, secondly, a more individual socialized behavior—ultrarapid motion pictures revealed to Dr. William A. Hunt, Connecticut College for Women, and Dr. Carney Landis, Columbia University.

Because of the way doses of glandular secretions change behavior, Dr. Roy G. Hoskins, Harvard University, proposed that "chemical conditioning" should be accepted as a new psychologic principle.

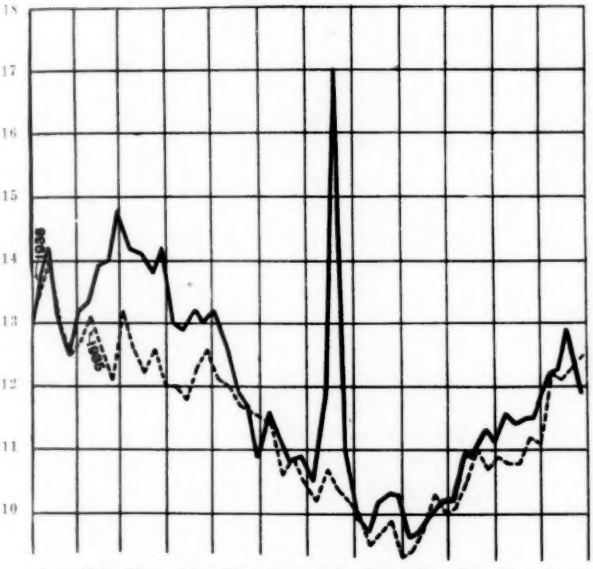
Chimpanzees can be taught to use gestures and vocalization to solicit aid from another animal in a cooperative task, Dr. Meredith P. Crawford, Yale University, found.

MAN'S SURVIVAL

The miracle of man's history is not modern science or invention, but the fact that human beings have been able to survive through thousands of years against terrific odds. Diphtheria, typhoid fever, smallpox, cholera, bubonic plague and yellow fever infested trade lanes and passed from village to village, killing appalling numbers of the defenceless. Man, with the aid of science and invention, has destroyed these enemies. He swarms over the earth. Now he plans to turn the weapons he used to increase his numbers, to the task of reducing them by war. Disease does a better job of improving the

race than war because it tends to weed out the weak, while war kills the strong. America's best allies in peace or war are not statesmen or diplomats, conferences or treaties, but the Atlantic and Pacific oceans.—*Public Relations Bureau, Medical Society of the State of New York.*

DEATH RATES PER 1,000 POPULATION OF CITIES REPORTING FOR 1936*



*Department of Commerce, Bureau of the Census.

GIFT OF THE GENERAL EDUCATION BOARD TO THE MEDICAL SCHOOL OF THE UNIVERSITY OF CHICAGO

The General Education Board of New York has given to the University of Chicago the sum of $3,000,000 to be spent for the development of the Medical School and the improvement of the University generally. This is the largest unrestricted gift ever received by the University.

Although the trustees have absolute discretion in spending the grant, the background of the discussions leading to the gift suggested that about $360,000 a year would go to medicine. Dr. Hutchins explained that the primary interest of the General Education Board in making the grant was in medicine. The Medical School was founded in 1927 with the help of the board, which since has been making temporary grants to assist the school. The present gift is intended to continue these grants and to provide a substantial sum for improvement.—*Science* **85**:39 (Jan. 8) 1937.

RECENT DEATHS

HOUSTON—John Alexander Houston, M.D., of 75 High Street, Northampton, Massachusetts, died at his home, January 18, 1937, after a short illness.

A native of Haverhill, Dr. Houston was graduated from Bowdoin Medical School in 1880. He practiced medicine for a few months in Winterport, Maine, and later, served as house physician in the Maine General Hospital in Portland. In 1882 he was appointed physician at the Worcester State Hospital and in 1889 became assistant superintendent of the Northampton State Hospital. In 1897, he was promoted to the superintendency of that institution, which position he held until his retirement in 1929. He was a retired member of the Massachusetts Medical Society.

His widow, Dr. Angela Bober Houston, and one son, John A. Houston, Jr., survive him.

PIERCE—Matthew Vassar Pierce, M.D., of 48 Centre Street, Milton, Massachusetts, died at his home on January 24, 1937. He was in his eighty-second year and had practiced medicine in Boston and its vicinity for more than half a century.

Born in Boston, Dr. Pierce was graduated from Harvard College in 1877 and from the Harvard Medical School in 1880. He then went to Germany for two more years of medical study, after which he returned to this country and set up practice in Milton.

He was a former president of the Norfolk District Medical Society and a member of the American Medical Association, the Dorchester Medical Club, the Harvard Club of Boston and the Milton Club.

A daughter, Mrs. George W. Benedict, Jr., of Cohasset, and two sons, Roger Pierce and Lincoln Pierce, both of Milton, survive him.

MATHER—John A. Mather, M.D. of Greenfield, Massachusetts, died suddenly at his home on January 16, 1937.

Born at Colrain in 1876, Dr. Mather was graduated from Baltimore Medical College in 1902. He practiced medicine at Colrain until 1920, when he opened offices in Greenfield. He was especially noted for his x-ray work and was roentgenologist at the Franklin County Hospital. During the World War he served in the medical corps and, in 1917, was a member of the medical advisory board of Greenfield.

He was a member of the Massachusetts Medical Society, the American Medical Association, and the Masonic Order.

His widow, Mrs. Gertrude Smith Mather, a son, Roland C. Mather of Providence, and a daughter, Miss Margaret M. Mather survive him.

REPORT OF MEETING

THE WACHUSETT MEDICAL IMPROVEMENT SOCIETY

A special meeting of the Wachusett Medical Improvement Society was held on November 24, 1936 at the Holden District Hospital. The meeting was called to order at 8:15 p. m. by Dr. Edward Crane, President of the Society.

Dr. Crane: This is an extra meeting of the Wachusett Medical Improvement Society to present the first

of a series of lectures on the parliamentary conduct of members at meetings. As a rule, the members lack the knowledge of the fundamental order of law and procedure at meetings. As Dr. Washburn said today, "It would be very hard to choose more than one or two members of the Worcester District Medical Society to the office of President of the State Society because of the lack of knowledge of Parliamentary law." Charles W. Proctor, Esq., of Holden and Worcester will proceed with his lecture to bring out the main points in regard to the parliamentary conduct of members at meetings.

MR. PROCTOR: *Mr. President and friends*—It is not so much my purpose tonight to present the method of making motions and voting as how to conduct one's self at meetings. I have considered this subject at some length and I know how difficult it is to make a speech or talk of this kind attractive and I shall attempt to do nothing more than to state my points clearly. The first question is how to organize meetings.

PARLIAMENTARY CONDUCT OF MEMBERS AT MEETINGS

Organization of Meeting

The members being assembled at the place and time appointed, one of them, without authority but usually by prearrangement, requests them to come to order. They thereupon seat themselves and he suggests the necessity of their being organized before proceeding to business and requests someone to nominate someone to act as chairman of the meeting. As soon as names are suggested, he puts the first one mentioned as nominated and puts the question whether he should be asked to preside. If decided in the affirmative, such person proceeds thereafter and if decided in the negative another is then called for until the choice is effected.

The next business is the election of the secretary which is made on nominations received by the new chairman. Such officers may be either temporary or permanent and if desired to have a more formal selection the next business is generally the appointment of the Nominating Committee upon whose report the meeting proceeds to organize itself, either accepting the report of the Committee or proceeding to make new selections of their own.

Committee on Credentials

Before proceeding to business when any members are elected as representatives of some other body or group it is essential to ascertain who are duly elected as members. After the temporary and before the permanent organization or after the permanent organization and before proceeding to other business a Committee on Credentials is appointed to approve the credentials and those who have been duly nominated, and to listen to the rival claims in case of dispute.

Right of Disputed Members to Be Present

When the question involving the right of a member to his seat arises any claimant for the seat is entitled to be heard by the Committee and by the meeting itself, but, as soon as he has been heard, it is his duty to withdraw from either of such bodies and remain away until the question is decided. If by the indulgence of the body he is permitted to remain he should neither participate in the discussion nor vote on the question, since to do so would be to permit him to act as judge in his own behalf.

Presence at Meetings

No person has a right to be present in a room where a meeting is being held without the consent of the assembly and consequently if the person refuses to withdraw or conducts himself in a disorderly or improper manner the assembly may employ sufficient force to eject him.

Procedure

After the chairman has assumed the chair and after the appointment of the secretary, the business is conducted on motion of the members. If a member desires to address the meeting or to make a motion, and so forth, it is his duty to rise, address the chairman and gain the recognition of the chair before proceeding further.

Quorum

The quorum consists of the number of members actually in attendance at a meeting and includes those not voting on a matter before the meeting as well as those who vote one way or the other. In the absence of By-Law or other rule the quorum consists of a majority of the members or delegates of the organization and this is true in all deliberative assemblies, meetings of committees, and so forth. Where a meeting is called informally without By-Laws, and so forth, the quorum is composed of those who attend the meeting. The By-Laws of all societies should provide for a quorum large enough to secure a fair representation of opinion and small enough so that it is easy to obtain under ordinary circumstances. Upon the failure to obtain a quorum nothing can be done except to try to get one, or to take a recess to some hour designated or to adjourn. Unanimous consent does not give permission to act in the absence of the quorum.

Should a meeting begin with a quorum present and should such number of members leave during the meeting as to reduce the number remaining below the number necessary to constitute a quorum the meeting cannot act further, except to take steps to procure the attendance of a sufficient number to be a quorum, to recess to a definite hour or to adjourn.

As previously stated, the By-Laws or Constitutions of societies should provide the number necessary to constitute a quorum and in case an amendment is desired care should be offered in amending the By-Laws so that the vote is taken on one question of altering to a new number the number necessary for a quorum as otherwise, after the number provided by the old By-Law has been stricken out, the meeting may find itself powerless to vote on the substituted number.

Enforcement of Rules

When a rule relative to the manner of proceeding is disregarded or infringed by a member or by the presiding officer each member has the right to take notice thereof and any one member may require that the presiding officer or other officer whose duty it is shall carry such rule into execution and the rule must be enforced at once, without debate or delay. The rule cannot be suspended after a member has asked for its enforcement until it has actually been enforced.

Sessions and Meetings of Bodies

A session embraces all the meetings of the body held until it is finally adjourned but a meeting lasts only until the members have left their seats by mutual consent. Where the By-Laws provide for regular meetings at stated intervals each meeting is a separate session, hence the motion to adjourn made at a meeting before the close of the session adjourns it only until the time assigned for the next meeting. If it is desired to break up the session altogether the motion should be to close the session or to adjourn without day. Rules passed for the conduct of the session continue throughout the session and during the several meetings.

Principles of Decision

Votes and elections are both decided on the principle of majority rule and that means the majority of those present and voting, either affirmatively or negatively, upon a subject. Members who do not vote are counted in determining the quorum but not counted either affirmatively or negatively in voting on a proposition. It is likewise the rule that in the election of officers, unless the By-Laws or other rules of proceedings provide otherwise, a person is not elected until he receives a majority of the votes cast and so if three people or more contest for an office, there is no election until the leading candidate has secured more votes than all those cast for the others combined.

Rules of Order

It is the duty of each member when engaged in a debate to confine himself within the rules of order and to observe decorum during the conduct of the meeting. Each member is equal with every other and has the same right with every other to submit his proposition to the assembly, to explain and recommend it in discussion and have it examined and deliberately decided by the body in which he belongs. It is his duty to conduct himself both in debate and in the assembly so as not to obstruct any other member in the enjoyment of his equal rights.

It may be stated generally that no member should disturb another or the assembly itself by hissing, coughing or spitting, by speaking to or whispering to other members, by standing up to interrupt others who are then engaged in debate, by passing beside the presiding officer and the member who is speaking, by leaving his seat and walking up and down or by taking books or papers from the secretary or from elsewhere in the hall or by writing. It is his duty to remove his hat when he enters the assembly and remain uncovered until he leaves the assembly hall. These rules apply whether the assembly is engaged in debate or not. Assaults, personal aspersions by one member on another, threats, challenges, and so forth, are a high breach of decorum in the assembly hall. In case of breach of decorum it is the duty of any member to complain to the meeting and to call its attention to the offender. It is particularly the duty of the presiding officer to do so and to call the offender by name and state the conduct of which he is guilty. In case he does so it is the assembly's privilege to suspend whatever business is then before it and take proper disciplinary measures for the correction of the offense and the punishment of the offender. The meeting may allow the offending person to remain and he is permitted to speak by way of excuse but not otherwise. It may, if it desires, require him to withdraw and remain absent during the discussion of his offense. If he remains present during the discussion he is disqualified from voting as he cannot be judge on his own case.

Punishments for disorderly conduct are reprimanding, exclusion from the assembly, prohibition to speak or vote for a specified time and expulsion to which may be added other forms of punishment as by apology, and so forth, as the assembly may see fit to impose and require the offender to submit under pain of expulsion or other discipline.

Order of Debate

The presiding officer should not participate in debate in any other capacity than as a presiding officer without leaving the chair. He is permitted, however, to state matters of fact within his own knowledge, to inform the meeting on points of order or the course of proceeding when called upon for that purpose and when he deems it wise to do so and if his rulings are appealed from he may address the assembly and give the reason for his ruling.

Manner of Speaking

As has been previously stated a member desiring to speak or to offer a motion, and so forth, should rise and address himself to the presiding officer and wait until he is recognized. The presiding officer should thereupon call him by name and if he is unfamiliar with his name ask it from the member who has risen, then call him by the name, telling him he has the floor, if necessary, and the member should then state what he has to say and sit down when he has finished his remarks.

If a member desires to make a motion he should state his motion and sit down and wait for a second. After his motion has been seconded he may again arise and it is the duty of the chair to recognize him in preference to others that have arisen at about the same time. If during the course of his speech a member is interrupted and yields the floor to another, even though he yields it for a particular

purpose, he has in fact yielded it for all time and the member who is now on the floor may speak to whatever question is before the meeting although he has violated his agreement with the member yielding the floor.

During debate no member should mention another member by name but should refer to him by his location in the meeting, by the order in which he has spoken or by some other means of identifying him. This is for the purpose of avoiding excitement of personal feeling either in favor of or against a particular man and to separate, as far as possible, the official from the personal character of each member.

If the presiding officer desires to speak, it is his duty to leave his place and sit with the other members at the meeting. He should not interrupt a member who is speaking but upon such member concluding his speech the presiding officer should arise and address the chair and should be recognized by the chair in preference to all others.

Matter in Speaking

A member who speaks in debate should confine himself to the question (and not speak impertinently or beside the subject) but so long as he does so he may speak as long as he pleases, and if his remarks become tiresome they cannot be interrupted, except, of course, that individual members may find means to bring them to a close.

A speaker should not use indecent language against the proceedings of the assembly or reflect upon any of its prior decisions unless he is speaking on a motion to rescind some prior decision but reflections on a proposition under consideration, or on the action of a committee that reported it, are not reflections against the assembly itself and are proper if otherwise unexceptionable.

Members should refrain from personalities and should not revile or slander one another and the motives of the person or persons proposing or opposing an action of the assembly are not open to question in debate. In general, whether a member's remarks are relevant or not is to be decided by the presiding officer but he should be slow to call a member to order unless the breach is obvious or unless the speaker otherwise clearly violates the principles of debate as already outlined.

While the question of motions has been left for a subsequent paper it often happens that a motion to amend, or lay on the table, or similar matters are pending. In such a case the member must confine his remarks to the particular motion rather than to the main question and remarks which relate to the main matter but not a matter which is at that moment under debate are as irrelevant as if they did not relate to any part of the question.

If a member is called to order by the presiding officer or by another member for irrelevancy the question may be raised as to whether he should be allowed to continue in the manner in which he was speaking and if voted in the negative the member is still allowed to proceed but must abandon the objectionable course of his remarks. If he persists, however, in the objectionable features of his remarks, the meeting, but not the presiding officer, may refuse him permission to continue at all.

Laws of Speaking

It is an elementary rule (although honored oftener in breach than observance) that no member shall speak more than once to the same question although the debate in question is protracted and continues through several days and although the member who desires to speak a second time has changed his opinion during the course of the debate. In enforcing this rule, however, it should be noted that there are often several questions which arise before a vote is taken such as on amendments, such as motions to lay on the table and other motions which may be called of a subsidiary nature, and the fact that a member has spoken to the main question or to some subsidiary question does not debar him from speaking to a subsidiary question or speaking each time a subsidiary question comes before the meeting. Strictly enforced, the rule above stated prevents a member from speaking a second time to a matter on which he has already spoken so long as other members desire to speak. When all have spoken who desired to speak for the first time such a member may speak a second or third time by leave of the meeting. He may also speak a second time in the same debate to clear up a matter of fact or to explain what he previously said but must keep within that line and not speak to the matter itself.

A member has no right to interrupt another member for the purpose of explaining what he previously stated, but must wait until the member has finished or may ask the member to yield for the purpose of his explanation, but if this is done the yielding member yields the floor altogether.

Stopping Debate

At any time during debate it is competent for a member to obtain the floor and move the previous question which if moved and seconded, precludes all further debate until the motion on the previous question has been disposed of. While this motion will be covered in the paper dealing with motions, yet, historically, an affirmative vote on this motion required the main question to be put at once and a negative vote decided that the main question could not be put at all. This is not the rule today and a negative vote on motion of the previous question means that debate may continue as if the motion had not been made.

It is also competent for the meeting, before a matter is taken up, to limit debate to a certain hour when all debates shall cease or to limit each speaker to a certain number of minutes.

Decorum in Debate

While a member is speaking no member should interrupt him, but if the speaker finds that he is not regarded with the respectful attention that his

equal right demands and that the meeting does not desire to hear him and in fact endeavors to drown his voice, his most prudent course is to sit down rather than to incur the displeasure of those who are guilty of such rudeness toward him, but that is a matter for the speaker to determine for himself and if he is willing to undergo the displeasure of those who are heckling him, that is his privilege. It is, of course, the duty of the presiding officer to support the member who is speaking, to call the others to order and even to name those who persist in disturbing him, but if after repeated trials he finds that the meeting will not support him in the exercise of his authority he is justified in allowing the meeting to proceed in its disorderly conduct.

Remedy for Offensive Language

If a member while speaking makes use of language personally offensive to another or insulting to the assembly, any member who thinks it proper may complain of it to the assembly in the following manner: The member speaking is interrupted by another member rising and calling to order. The member who objects or complains of the words is then called upon by the presiding officer to state the words which he complains of, repeating them exactly as he conceives them to be spoken in order that they may be written by the clerk or the member may proceed at once to state the words, either verbally or in writing, and desire that the clerk take them down at the table. The presiding officer may then direct the clerk to take them down in the form and manner in which they are stated by the member who objects. This is discretionary with the presiding officer. The words being thus written down and forming a part of the minutes in the Clerk's book they are next to be read to the member who was speaking who may deny they are the words that he spoke, in which case the members decide whether they were or were not or may amend the words so as to conform to what was said. If they are not denied or are found by the assembly to be true, then the member who used them may either justify them or explain the sense in which he used them so as to remove the objection of their being disorderly, or he may apologize for them. If the justification, explanation or apology is deemed sufficient no further proceeding is necessary and the member may resume and go on with his speech, but if any two members making and seconding the motion think it necessary to state a question so as to take the sense of the meeting on the words and whether the member in using them has been guilty of any offense toward the assembly the speaker must withdraw before that question is stated and the sense of the assembly must be taken with such further proceedings in relation to the punishing of the member as may be thought necessary and proper. If no notice is taken of the words or of the speaker but the member is allowed to finish his speech and then any other business intervenes before notice is taken of the words, the words are not to be written down, nor can the member using them be censured. This is to prevent mistakes which must necessarily happen if the words complained of are not promptly reduced to writing.

DISCUSSION

MR. PROCTOR: That covers all I have to say tonight. I intended to take up motions at another meeting, and now I shall be glad to answer any question you may want to ask concerning the parliamentary conduct of members at meetings.

MEMBER: I would like to know if any other officer besides the presiding officer can make a motion or discuss a motion—such as the secretary.

MR. PROCTOR: Any officer, excluding the presiding officer, has the privilege to make a motion.

MEMBER: Why cannot a member making a motion explain his motion beforehand?

MR. PROCTOR: Because it is better to have the meeting informed as to what is before it.

MEMBER: Who presides when the presiding officer takes the floor?

MR. PROCTOR: He may give the chair to anyone he wishes. Usually he gives it to the Secretary, who is sitting near him, but it is not compulsory for him to do so.

MEMBER: Will you please define "previous question"?

MR. PROCTOR: When a man rises in his seat and and says, "I move that the main question be put", it is the duty of the chair to say "The question is now put," and if the vote is in the negative the main question is "put" in the affirmative.

MEMBER: Then the previous question and the main question are not the same thing?

MR. PROCTOR: No, the main question is still open for discussion while the previous question is the one put into effect at the present time.

MEMBER: In what way may there be more than one motion before the meeting?

MR. PROCTOR: For instance, I may move that the meeting be held at Putnam & Thurston's on Wednesday. Someone else may move that it be held Tuesday instead of Wednesday as a way of amendment.

MEMBER: Does the presiding officer attempt to keep any notes or does he rely on the secretary to write down the lines?

MR. PROCTOR: It is a breach of etiquette for him to take notes. If he cannot remember the lines he must rely on the secretary to state them.

MEMBER: Does the presiding officer ever preside seated in his chair?

MR. PROCTOR: It is not proper for him to do so, but if someone speaks any length of time he often sits down in his chair.

MEMBER: Must he stand while the debate is going on?

MR. PROCTOR: He should do so.

MEMBER: Has a member any redress if the chair fails to recognize him?

MR. PROCTOR: Yes, the member may appeal from

the ruling of the chair in recognizing one member instead of another.

MEMBER: I have noticed that, when a motion has been made and seconded, the motion is often repeated by the presiding officer. Is the repetition of a motion proper?

MR. PROCTOR: That is a matter of option for the chair. It is considered good practice for the chair to do so.

MEMBER: What happens if the chair simply refuses to recognize the man who is up first?

MR. PROCTOR: He can appeal from the ruling of the chair.

MEMBER: And the chair is forced to recognize him?

MR. PROCTOR: Yes.

MEMBER: *Mr. President*—I do not think we all realize the value of this talk, so I would like to ask if it would not be possible to put a copy of this lecture in the hands of every member of the Medical Society. I would like a copy of it myself.

MEMBER: In reply to the question which Dr. Davidson asked, I can say that I received a letter from an editor asking that a stenographic copy be sent to him, but whether that means that he will publish it I do not know. However, I am sure that we can obtain reprints of it.

MR. PROCTOR: I wrote my manuscript and anyone is perfectly welcome to it.

MEMBER: But that does not answer my question.

MEMBER: I don't think we can decide that question tonight.

MEMBER: I move this be made a regular meeting.

MEMBER: The motion made has not been seconded, but the ruling brings to mind the question—is it proper to say, I move you?

MR. PROCTOR: It is not a frequent use but it is proper.

PRESIDENT: Do I hear it seconded?

MEMBER: I second it.

PRESIDENT: It has been moved and seconded. Are there any comments upon the motion?

MEMBER: Would this be a proper procedure?

MR. PROCTOR: I do not think so.

MEMBER: The By-Laws provide that our meetings be held once a month, and one is coming, so this is not a regular meeting.

PRESIDENT: We are trying to come to some sort of understanding about parliamentary law, and we should say that we cannot call it a true meeting unless it is so specified before holding the meeting.

MEMBER: I suppose it is proper for me to withdraw my motion?

MR. PROCTOR: Yes.

PRESIDENT: I wish to thank Mr. Proctor for coming here tonight, and we should like to have him here at the next meeting.

The meeting was adjourned at 9:25 p m.

NOTICES

REMOVALS

Doctors FRANKLIN G. BALCH, FRANKLIN G. BALCH, JR., WALTER E. GARREY, WILLIAM W. HOWELL, HORACE K. SOWLES and EDWARD L. YOUNG, JR., have moved their offices from 279 Clarendon Street to 330 Dartmouth Street, Boston.

CONFERENCE OF CLINICAL PATHOLOGY

A monthly conference of clinical pathology will be held at the Boston City Hospital on Wednesday, February 10, at 12 o'clock noon, in the Pathological Amphitheater.

JOSEPH E. HALLISEY, M.D.,
Secretary Medical Staff.

LAWRENCE CANCER CLINIC

Established April 17, 1928
Lawrence General Hospital
Lawrence, Massachusetts.
February 10, 1937.

To the Physicians of the North Half of Essex County:

Dear Doctor:

The regular Lawrence Cancer Clinic, to be held at Lawrence General Hospital, 1 Garden Street, Lawrence, upon Tuesday, February 16, at 10:00 a. m., will be a Demonstration and Teaching Clinic for physicians, with Channing C. Simmons, M.D., of Boston, Associate in Surgery in the Graduate Courses in Medicine at Harvard University Medical School, Surgeon-in-Chief to Collis P. Huntington Memorial Hospital, member of the Cancer Commission of Harvard University, and Visiting Surgeon to the Massachusetts General Hospital, present as consultant. You are invited to accompany any of your patients whom you desire shall have this service, or to send them with a note, and a report will be returned to you. This service is gratis. Your attendance at the Clinic is always welcome.

This clinic is endorsed by the Committee on Postgraduate Instruction of the Massachusetts Medical Society.

Committee
ROY V. BAKETEL, M.D.,
CHAS. J. BURGESS, M.D.,
JOHN J. MCARDLE, M.D.,
HARRY H. NEVERS, M.D.,
THOS. V. UNIAC, M.D.,
J. FORREST BURNHAM, M.D.,
Chairman.

MEDICAL CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 p. m. on Thursday, February 11, in the Amphitheater of the Peter Bent Brigham Hospital, Dr. G. Philip Grabfield, Associate in Pharmacology, Harvard Medical School, and Associate in Medicine, Peter Bent Brigham Hospital, will give a medical clinic. To it are cordially invited practitioners and medical students.

AWARDS FOR RESEARCH IN PSYCHIATRY

The New England Society of Psychiatry, at its next spring meeting, will make two awards, one of $50 and one of $25, to the writer (or writers) of the best papers completed or published during the calendar year of 1936 embodying research in psychiatry by a younger worker (or workers). Physicians, psychologists, social workers and others are eligible. Membership in the Society is not a requisite.

Authors, who present articles for consideration, should make arrangements with the publisher of their paper to preserve the plates, until such time as the awards are made, from which reprints could be furnished to the members by the Society.

Writers who have once received an award are not again eligible. Seasoned writers, senior physicians and heads of departments in which there are junior workers, while not inevitably excluded, will not generally be regarded as eligible for the awards.

The work on which the papers are based should preferably have been done in New England or by workers now living in New England.

The papers will be examined by a committee of three members that are accustomed to reviewing papers and by the Executive Committee of the Society. They will be judged on the basis of their scientific quality.

Copies of articles or marked copies of journals in which the articles have appeared should be sent before March 1, 1937, to the Secretary of the Society.

Superintendents of institutions, public or private, for the care of mental patients in New England, also deans of colleges and universities maintaining medical schools or departments of psychology, are requested to post a copy of this notice, and to send to the Society a list of such papers published as they think entitled to be considered for the awards.

HARLAN L. PAINE, M.D., *Secretary.*

North Grafton, Mass.

LOWELL INSTITUTE LECTURES

Lewis W. Hackett, M.D., Dr.P.H., assistant director of the International Health Division of the Rockefeller Foundation and representative of the Rockefeller Foundation in Italy and Albania, is giving the current series of free public lectures sponsored by the Lowell Institute. The main topic is "Man Against Malaria in Southern Europe," and the individual titles are: (1) "The World War and Malaria"; (2) "The Malaria Puzzle in Europe"; (3) "The Solution of the Problem"; (4) "Modern Strategy in Malaria Control"; (5) "The Albanian Experiments" and (6) "Land Reclamation in Italy."

The lectures are being given Tuesdays and Fridays at 8:00 p. m. in Huntington Hall, 491 Boylston Street, the first lecture having been given on February 2.

MEMORIAL MEETING FOR DR. FRANKWOOD WILLIAMS

A memorial meeting for Dr. Frankwood E. Williams will be held on Friday evening, February 5, at 8 o'clock at the Beth Israel Hospital Auditorium, 330 Brookline Avenue, Boston. The speakers will be Herbert C. Parsons, Chairman; Dr. Douglas A. Thom, "Frankwood Williams — Psychiatrist and Man"; and Miss Bertha Reynolds, "The Significance of Frankwood Williams's Work".

This meeting is sponsored by the American Association of Social Workers, Boston Chapter; Boston Council of Social Agencies; Inter-Professional Association, Boston Chapter; Massachusetts Society for Mental Hygiene; New England District, American Association of Mental Social Workers; and the New England Round Table on Psychiatric Social Work.

WILLIAM HARVEY SOCIETY

The next meeting of the William Harvey Society will be held Friday, February 12, in the Auditorium of the Beth Israel Hospital, Boston, at 8:00 p. m.

PROGRAM

Speaker: Dr. Richard C. Cabot, Professor of Clinical Medicine, Harvard University Medical School.

Subject: The Wisdom of the Body.

Chairman: Dr. A. Warren Stearns, Dean of Tufts College Medical School.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheater (Shattuck Street Entrance), Tuesday evening, February 9, at 8:15 p. m.

PROGRAM

Presentation of Cases.

"Intestinal Obstruction—the Special Importance of Potassium." John Scudder, M.D., Fellow in Surgery, Presbyterian Hospital, New York, N. Y.

Medical students and physicians are cordially invited to attend.

MARSHALL N. FULTON, M.D., *Secretary.*

NEW ENGLAND HEART ASSOCIATION

The next meeting of the New England Heart Association will be held at the Beth Israel Hospital, Boston, Mass., Monday, February 8, 1937 at 8:15 p. m.

PROGRAM

1. Pathologic Physiology of the Circulation in Tricuspid Stenosis and Insufficiency. Case presentation. Dr. Mark D. Altschule.
2. The Clinical Significance of the Sedimentation Rate in Angina Pectoris and Coronary Thrombosis. Dr. Morton G. Brown.
3. Observations on Cardiac Hypertrophy. Dr. David Davis.

4. Duration of Attacks of Angina Pectoris and the Effect of Nitroglycerin and Amyl Nitrite in Terminating the Attacks. Dr. J. E. F. Riseman.
5. The Effect on the Cardiovascular System of Fluids Administered Intravenously. Dr. R. Gilligan.
6. Complete Occlusion of Coronary Artery With and Without Cardiac Infarction. Dr. M. J. Schlesinger.
7. Experimental Studies on the Effect of Temporary Occlusion of Coronary Arteries. Dr. H. L. Blumgart.

All members of the New England Heart Association and interested physicians are invited to attend.

JAMES M. FAULKNER, M.D., *Secretary.*

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, FEBRUARY 8, 1937

Monday, February 8—

*8:15 p. m. New England Heart Association. Beth Israel Hospital, Boston.

Tuesday, February 9—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Anemia in Infants. Dr. James M. Baty.

9:30 a. m. Massachusetts General Hospital. Thoracic Clinic. Ether Dome.

*8:15 p. m. Harvard Medical Society. Peter Bent Brigham Hospital Amphitheater (Shattuck Street entrance).

Wednesday, February 10—

8 a. m. Massachusetts General Hospital. Grand Rounds. Orthopedic Department.

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Hospital Case Presentation. Dr. H. G. Brugsch.

12 M. Boston City Hospital, Conference of Clinical Pathology, Pathological Amphitheater.

†12 m. Clinicopathological Conference. Children's Hospital Amphitheater.

4 p. m. - 5 p. m. Surgical Pathological Conference. Dr. Cutler and Dr. Wolbach, Peter Bent Brigham Hospital.

Thursday, February 11—

*8:30-9:30 a. m. Exchange visit, Surgical and Orthopedic Staffs of the Peter Bent Brigham and the Children's Hospitals, held this week at the Children's Hospital.

9 a. m. Massachusetts General Hospital. Surgical Grand Rounds.

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Clinicopathological Reports on Outpatients. Dr. Joseph H. Kaplan.

9:15 a. m. Massachusetts General Hospital. Neurological Conference. Ether Dome.

12 m. Massachusetts General Hospital. Clinicopathological Conference.

*3:30 p. m. Medical Clinic at the Peter Bent Brigham Hospital. Dr. G. Philip Grabfield.

Friday, February 12—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Acid-Base Patterns in Health and Disease. Dr. John H. Talbott.

10 a. m. Massachusetts General Hospital. Fracture Rounds.

8 p. m. William Harvey Society. Beth Israel Hospital Auditorium, Boston.

Saturday, February 13—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Hospital Case Presentation. Dr. Jacob Schloss.

*10 a. m. - 12 m. Staff Rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

8 p. m. Massachusetts Psychiatric Society. Boston Psychopathic Hospital.

Sunday, February 14—

4 p. m. Free public lecture at the Harvard Medical School, Building D, Longwood Avenue, Boston. "Surgical Aid in Lung Diseases." Dr. Edward D. Churchill.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

February 4—Faulkner Hospital Clinical Meeting at 5 p. m.

February 5—Boston Pathological Society, 8 p. m. Massachusetts General Hospital, Pathology Department.

February 5—Memorial Meeting for Dr. Frankwood Williams. See page 229.

February 8—New England Heart Association. See page 229.

February 9—Harvard Medical Society. See page 229.

February 10—Boston City Hospital, Conference of Clinical Pathology. See page 228.

February 11—Medical Clinic at the Peter Bent Brigham Hospital. See page 228.

February 11—Pentucket Association of Physicians. Hotel Bartlett, 95 Main Street, Haverhill, at 8:30 p. m.

February 12—William Harvey Society. See page 229.

February 13—Massachusetts Psychiatric Society, 8 p. m. Boston Psychopathic Hospital.

February 16—Lawrence Cancer Clinic. See page 228.

February 25, 26, 27—The New England Hospital Association. Hotel Statler, Boston.

March 6, June 8 and 9—American Board of Obstetrics and Gynecology. See page 78, issue of January 14.

March 30 - April 2—First International Conference on Fever Therapy. Postponement notice. See page 52, issue of July 2.

April 12-16—The Postgraduate Institute of the Philadelphia County Medical Society. See page 185, issue of January 28.

April 21-24—American Society for Experimental Pathology. See page 1075, issue of May 21.

July 6-11—Fifth International Congress of Hospitals. See page 37, issue of January 7.

October 5-8—American Public Health Association Meeting. New York City.

October 25-29—American College of Surgeons. Chicago, Illinois.

DISTRICT MEDICAL SOCIETIES

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

February 10—See page 190, issue of January 28.

FRANKLIN DISTRICT MEDICAL SOCIETY

Will meet at the Weldon in Greenfield at 11 a. m. the second Tuesdays of March and May.

CHARLES MOLINE, M.D., Secretary.
Sunderland.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

March 16—Danvers State Hospital, Danvers.
May 11—Bear Hill Golf Club, Stoneham.

KENNETH L. MACLACHLAN, M.D., Secretary.
1 Bellevue Avenue, Melrose.

NORFOLK DISTRICT MEDICAL SOCIETY

February 16—See page 138, issue of January 21.

March 30—8:15 p. m. New England Deaconess Hospital. A Symposium on Diabetes entitled "A Survey of the Diabetic Work of the George F. Baker Clinic in the New England Deaconess Hospital." Communications and Case Presentations by the Staff. Drs. Elliott P. Joslin, Howard F. Root, Priscilla White, Alexander Marble and Allen P. Joslin.

May—Annual Meeting. Details to be announced.

Note: The Censors will meet for the examination of candidates on the first Thursday of May. Fee of $10.00 is payable at the time of examination. Application blanks may be obtained by writing the Secretary, furnishing name, address and name of school of graduation in medicine. Application must be made at least three weeks prior to date of examination. Candidates whose applications are on file will receive proper notices.

FRANK S. CRUICKSHANK, M.D., Secretary.
1247 Beacon Street, Brookline.

PLYMOUTH DISTRICT MEDICAL SOCIETY

March 18—11 a. m. Brockton Hospital.
April 15—Annual Meeting. 11 a. m. Ducy Hospital.
May 20—11 a. m. Lakeville State Sanatorium.

FRED F. WEINER, M.D., Secretary.
231 Main Street, Brockton.

SUFFOLK DISTRICT MEDICAL SOCIETY

March 31—Boston Medical Library. 8:15 p. m. "Social Insurance—It Affects the Medical Profession." Dr. Charles E. Mongan. Discussion: Dr. Channing Frothingham.

April 28—Annual Meeting. Boston Medical Library. 8:15 p. m. "Problems in Surgical Diagnosis." Dr. Howard M. Clute.

CONRAD WESSELHOEFT, M.D., President,
CHARLES C. LUND, M.D., Secretary.

WORCESTER DISTRICT MEDICAL SOCIETY

February 10—Worcester State Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

March 10—The Memorial Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

April 14—Worcester Hahnemann Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

May 6—At 4:30 in the rooms of the Worcester Medical Library, Inc., at 34 Elm Street, Worcester, will be held the spring meeting of the Board of Censors.

Wednesday Afternoon and Evening, May 12—Annual Meeting. Time and place for this meeting will be announced in an early spring issue of the Journal.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester.

BOOK REVIEWS

Psychiatry for Practitioners. Oxford Medical Publications. By Various Authors. Edited by Henry A. Christian. 646 pp. New York: Oxford University Press. $6.50.

This book is reprinted from Oxford Loose-Leaf Medicine and is a duplicate of Oxford Loose-Leaf Medicine, Vol. vii, already reviewed in this *Journal* 216:88 (Jan. 14) 1937.

Tissue Immunity. Reuben L. Kahn. 707 pp. Springfield and Baltimore: Charles C Thomas. $7.50.

This most unusual and interesting book consists, chiefly, of the listing of experiments concerning tissue immunity that were devised and carried out by the author and his assistants. Fifteen chapters contain experiments designed, in logical sequence, to determine the condition of the tissues in various states, such as nonimmune, immune, hyperimmune, disimmune (desensitized) and so forth, and each is concluded with a summary of the findings and a discussion of the clinical applications. These are followed by a chapter that lists forty-two hypotheses concerning the defense of the host against microorganisms and against protein antigens, and the final chapter deals with the practical aspects of tissue immunity.

He maintains that immunologic response is a *physiologic* function of the body and that it is evidenced by the ability of tissues to anchor or localize microorganisms or foreign proteins. The inflammatory reaction following parenteral injection of antigen is evidence of such immunity, and the intensity of the reaction is directly proportional to the degree of immunity. Allergy is merely hyperimmunity. He believes that immunity concerns, primarily, the ability of the cells in the tissues or of the mobile phagocytes to combine with antigen and that circulating antibodies, with the exception of antitoxins, are unnecessary and merely represent an excess of immune globulins. Some tissues in actively immunized animals, chiefly the skin and subcutaneous tissues, develop a greater degree of antigen-combining capacity, hence, of defense, than others; this is not true of passively immunized animals. He asserts that necrosis presumes the presence of an excess of antigen in an existing inflammatory lesion in an immune animal, brought about either by an excess of circulating antigen, by residual, uncombined antigen in the lesion or by the reinjection of antigen into the lesion, and that such necrosis is indicative of aggressive action of the antigen against the host.

Undoubtedly the author will be criticized for his disregard of the humoral and phagocytic aspects of immunity, of the possible primary toxicity of certain of his bacterial antigens and of the possible mechanical differences in tissue absorption due to anatomic structures and for his interpretations of observed experimental facts. Nevertheless, he has furnished a fascinating concept of immunity that logically follows detailed experimental work and that satisfactorily explains many puzzling immunologic problems. The book should be of exceptional interest to laboratory workers concerned in the study of bacteriologic, serologic and immunologic problems, to physicians particularly interested in infectious diseases and the so-called "allergic states" and to public health officers in general.

A Text-Book of Neuro-Anatomy. Albert Kuntz. Second Edition, Thoroughly Revised. 519 pp. Philadelphia: Lea & Febiger. $6.00.

The first edition of this book was highly recommended in a review appearing in *The New England Journal of Medicine*, Nov. 12, 1931. It now appears in a larger and more useful form; the 359 pages of the earlier edition have grown to 519 and three new chapters have been added. As an anatomy, the opinion given in regard to the first edition still holds good in relation to the second. The author, however, has failed to advance with the times and much that is needed by the modern neuroanatomist and physiologist is absent. There is no description, for instance, of Vogt's numerical designations of the cerebral cortex, used as a reference in practically all the current literature. The author, moreover, gives no account, in his chapter on the function of the cerebral cortex, of the recent researches in physiology carried on by Fulton, Dusser de Barenne, Bard, Jacobsen and others and the clinical studies of Brickner, Penfield, Foerster and their colleagues. Thus the book, a sound but rather old-fashioned type of textbook, lacks much that would be of use to the research student.

Surgical Emergencies in Children. Harold Clifford Edwards. 274 pp. Baltimore: William Wood & Company. $4.50.

This compact volume contains much that is of value to the general surgeon, the pediatrician and

the general practitioner. The author states clearly and briefly the important points in differential diagnosis of surgical emergencies in this age group. He also emphasizes the importance of early recognition of these diagnostic points by the general practitioner or pediatrician.

Of especial value are his chapters on the abdominal emergencies. The subject is well covered, clear and with sound reasoning. He advises radicalism when indicated, and conservatism when indicated. His views will coincide, to a large measure, with opinion in this country on these matters.

The chapters dealing with head injuries, fractures, pyogenic infections and surgical diseases of the thorax are also good. Here again he writes with open-minded conservatism, but stresses the importance of being radical when radical measures are needed.

We would disagree to some measure with his remarks on treatment of acute osteomyelitis in this age group. We feel that there is need for even greater conservatism than is at present taught in some communities in the treatment of early acute fulminating cases of osteomyelitis. Too early and too radical surgery is oftentimes to be avoided. We would take definite exception to the giving of antitetanic serum into the subarachnoid space, and would also place more reliance on sedatives such as avertin or luminal to control the spasm rather than the giving of chloroform for an hour or more as he advises.

The chapter on general anesthesia by Dr. Vernon Hall and the chapter on ear, nose and throat by Geoffrey H. Bateman reflect the English point of view, but there is little to criticize as most points are debatable largely from a personal point of view rather than any variance from generally accepted procedures.

The volume should prove a useful book for surgeons and medical men who have to deal with the age group considered.

The Therapeutic Agents of the Pyrrole and Pyridine Group. Including the Tropinol, Scopoline, Ecgonine, and Granatoline Derivatives. The Relation between Their Chemical Constitution and Pharmacologic Action. W. F. von Oettingen. 258 pp. Ann Arbor: Edwards Brothers, Inc. $4.75.

This volume is a continuation of a monograph previously published by the American Chemical Society under the title "The Therapeutic Agents of the Quinoline Group, the Relation between Their Chemical Constitution and Pharmacologic Action". It discusses the rather complex organicochemical structure of pharmacologically important derivatives of pyrrole and of pyridine. The systematic treatment of the various compounds involved savors of the type of reasoning by which Ehrlich arrived at his classical 606th compound. In short, in this volume organic structural chemistry joins hands with therapeutics in an effort to systematize biologic results on a chemical basis.

Among the types of substances discussed are many which are familiar to the general practitioner. Among them are nicotine, physostigmine, uroselectan or iopax, coramin, lobeline, beta-eucaine, atropine and hyoscyamine, homatropine, scopolamine, cocaine, and pelletierine. There are innumerable graphic formulas illustrating the chemical structure of these various compounds and their relation to one another. Clinical responses are mentioned along with methods of assay and physiologic effects observed in animal experiments.

On the whole, this is not a book which the busy practitioner of medicine will read throughout, although it might well appear in his study bookcase. Rather, it is a scholarly treatise to be used for reference in departments of pharmacology or of pharmaceutical chemistry, and it should be on hand in every up-to-date school of medicine or pharmacy.

The material is systematically arranged and clearly presented. There are many useful summarizing tables and illustrative charts. At the end there are some two-score pages of bibliography, and an excellent index.

A word of praise is due the publishers for the excellent reproduction of this work, which is photolithographed from perfect type-script. The many chemical formulas have been clearly printed at a cost much lower than one would have expected for this type of material.

The Foot. Norman C. Lake. 330 pp. Baltimore: William Wood & Company. $4.50.

The author offers in this book an excellent presentation of the evolution of the foot, its anatomy and physiology. He deals ably with the general care of the foot. He presents less satisfactorily many disorders common to the foot, and suggests appropriate treatment for each.

As in any book on a specialized subject the author offers his own personal views. The success or failure of such a work depends obviously on whether he succeeds in convincing the reader that his views are the popular ideas of the moment. In this book, treatment of the many disorders described must be very general to be condensed into 330 pages. Actual, constructive details of individual treatment are lacking. Reference to disorders, such as arthritis, of which the foot manifestations are a minor part of a systemic disturbance, are particularly unsatisfactory. The index would be more helpful to the reading practitioner seeking information quickly if some of the common infections, "athlete's foot," for instance, were listed by name rather than hidden under the general heading, "infections, focal". By and large, the book deals with those disorders of the feet which come under the realm of surgery, but no extensive orthopedic surgical operation is described in sufficient detail to be actually followed in practice. On the whole, the book is recommended "luke-warmly".